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TRANSLATOR'S PREFACE

THIS volume presents to the practitioner and to the student the problems in diagnosis which confront them at the bedside. In order to achieve this object, theoretical classifications are not adhered to; instead, diseases are grouped according to similarity of symptoms and points of general resemblance—considerations which in practice render their differentiation difficult. In this way the advantages of clinical teaching are most nearly attained, and by the presentation of a large number of cases, the value of this arrangement is further enhanced. The fragmentary and disjointed instruction which clinical demonstration necessarily entails, even under the most favorable conditions, is thus circumvented. Moreover, whenever feasible, the cases reported are followed to the operating table, at times to autopsy, to confirm or to correct the diagnosis.

In marked contrast to the numerous works on medical diagnosis now accessible to the American reader, works on surgical diagnosis are comparatively few; [illegible] the translation of Albert's book can fill this gap, it will fulfil its purpose.

ROBERT T. FRANK.

NEW YORK.

THE DIAGNOSIS OF
SURGICAL DISEASES

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BY

DR. E. ALBERT

LATE DIRECTOR AND PROFESSOR OF THE FIRST SURGICAL CLINIC
AT THE UNIVERSITY OF VIENNA

AUTHORIZED TRANSLATION FROM THE EIGHTH
ENLARGED AND REVISED EDITION

BY

ROBERT T. FRANK, A.M., M.D.

WITH FIFTY-THREE ILLUSTRATIONS

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COOPER MEDICAL
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THE DIAGNOSIS OF SURGICAL DISEASES

CHAPTER I

THE CAUSES OF ABNORMAL POSITIONS OF THE HEAD

THE mechanism by which the head is maintained in its physiological position has been studied by the Weber brothers. The results of their experiments, which were performed upon the cadaver, are very striking. A head severed from the rest of the body by a cut extending through the occipito-atlantal articulation, and resting solely upon the condyles, was placed upon a thin vertical cylinder. The head remained in this position of equilibrium without further support. It follows that there is at least *one* position of the head, and that one of labile equilibrium, in which it remains balanced without the intervention of muscular exertion or of ligamentous support.

In this position the head is directed straight forward, with the chin tilted slightly up. Physical considerations show that in this posture the centre of gravity of the head lies slightly in front of its condylar support, and therefore, that a slight downward force, applied behind the fulcrum, is able to keep the head at rest. It is evident that this office is performed by the muscles at the nape of the neck which keep the head properly balanced in all positions the body may assume. Observation has shown that in high degrees of weakness of this group of muscles the head falls forward.

In the clinic of my instructor, v. Dumreicher, I observed a girl whose head had sunk so far forward that it finally rested upon the sternum. The chin had caused a pressure-sore at this point. Constant sewing in a stooping posture had brought about the deformity. Examination failed to show any abnormality of the cervical vertebræ, but the muscles of the neck had almost disappeared from atrophy of disuse. The two rounded welts, normally formed by the muscles on either side of the spinous processes, were entirely wanting. The head was cautiously raised without encountering any resistance, but movement caused some uneasiness to the patient. Passive movement of the head and of the cervical segment was normal; the raised head, however, acted like a dead weight when left to shift for itself, and threatened to topple forward. Sudden straightening produced a painful degree of tension in the structures of the anterior region of the neck, therefore the correction was undertaken slowly and by degrees. After the head had been properly raised, it was maintained in position by means of a Glisson's sling attached to a jury-mast. Daily application of electricity was then resorted to, until the muscles had regained sufficient strength to support the head without artificial aid.

An analogous case was treated by v. Dumreicher. The patient was a man far advanced in years. His head had sunk downward and forward on to the sternum as the result of a slowly advancing weakness of the muscles of the neck.

These two cases are the simplest examples of abnormal position, and are directly correlated to the simple physiological facts just mentioned.

By what mechanism is the head moved? We distinguish three different motions:

1. Nodding movements occurring in the occipito-atlantal articulation.

2. Movements of rotation about the odontoid process of the axis.

3. Movements of flexion and extension, which result from the bow-shaped curving of the entire cervical segment.

Movements through a greater arc than that permitted by the atlanto-axial articulation are executed by means of the slight degree of rotation which takes place in the joints of the remaining vertebræ of the cervical portion of the spine. If the head is turned, with the cervical spine held fixed, a considerable degree of motion is possible; but still greater rotation can be effected by allowing the whole cervical segment to participate, not, however, without producing a painful feeling of tension. In order to look backward, the dorsal segment must also take part in the movement.

Nodding movements (extension and flexion) of the head do not occur to a considerable degree in any joints except the occipito-atlantal. To attain a still wider range, the aggregate obtained by bending the other cervical vertebræ either backward or forward is brought into play.

These various motions are limited, in the normal state, by the ligaments of the joints. Any force which carries the range of motion beyond these normal limits can do so only at the expense of the ligaments, which are consequently stretched or torn. As a result, we find *Diastases* of the vertebræ (rupture of the connecting ligaments, without displacement) and DISLOCATIONS (rupture with displacement of the bones).

At times the force is exerted either in such a fashion, or is met by such resistance, that the ligaments escape, but the vertebræ are torn and fracture results.

The complicated structure and the high degree of mobility of the cervical spine readily account for the combination of fractures, dislocations, and diastases so often met with in practice.

In the cervical segment of the spine we recognise the following varieties of dislocation:

1. *Luxatio capitis:* dislocation occurring between the head and atlas. Only two cases are on record. In the one case, in which the patient survived the accident five months, the head remained firmly fixed, with the chin resting upon the sternum and the vertex directed forward.

2. *Dislocation of the atlas:* a single indisputable case recorded. The atlas had rotated about the axis in such a fashion that the ligaments of its descending articular process on one side were completely ruptured, those of the opposite partially torn.

3. *Dislocation occurring between the other cervical vertebræ.*

(*a*) Bilateral, (*b*) unilateral dislocations.

Bilateral dislocations may be of two kinds: displacement of both processes in the *same* direction, that is, both right and left, either forward or backward (in which the upper vertebra is regarded as the dislocated bone). Displacement in opposite directions, in which the articulation of the right side moves forward, that of the left side backward, or vice versa. In this case the vertebra turns about its vertical axis, and in doing so ruptures the capsules of both its articular processes. The first variety is the result of a force which *pushes* the vertebræ apart, the second results from torsion.

Unilateral dislocations are complete or incomplete, depending upon the extent of displacement of the one articular process upon the corresponding articular surface of the vertebra below. The dislocation is complete if the process rests in the notch situated in front of the lower articular process; incomplete if it merely glides forward to the most anterior portion of the joint surface (subluxation).

By what signs may the above-mentioned accidents be recognised?

Let us consider the symptoms of a complete *bilateral* dislocation produced by a force *pushing* forward from behind. In the cervical spine the surfaces of the superior and inferior articular processes correspond to a plane directed from behind and below in a forward and upward direction. It follows that the dislocating force must move the upper (dislocated) vertebra forward and upward until it has passed over the highest, and, at the same time, most anterior portion of the articular surface of the vertebra below. The dislocated vertebra then slides down and rests in front of this process. In two cases reported by Ph. v. Walter the head occupied such a position that the occiput rested in the space normally found between the shoulders. From the extent and nature of such injuries, it is self-evident that the remaining ligaments which connect the vertebræ must also be torn, and the spinal canal so encroached upon that the cord is crushed, causing paresis of the parts below the lesion. The break in the continuity of the line normally formed by the transverse processes is the diagnostic sign we look for, and if we can convince ourselves that the transverse processes above a certain point are situated in front of those immediately below, no further uncertainty should be entertained.

In *bilateral* dislocation caused by *torsion*, the rotation of the head naturally forms the most prominent symptom. The picture presented does not, however, differ much from that due to a unilateral dislocation by torsion, in which only *one* capsule is ruptured, the other stretched, whereas in a bilateral dislocation of this variety *both* capsules are torn. It will not be necessary to consider both; discussion of a unilateral dislocation will suffice.

In order to understand the picture presented by a unilateral dislocation due to torsion, it must be borne in mind that, while the surface of the descending articular process of the superior vertebra is moving upward on the superior articular process of the subjacent vertebræ on the left side in these rotatory motions, it must move downward on the right side. If the motions are carried beyond the physiological limits the descending articular process of the superior vertebra on the left side will surmount the superior articular process of the vertebra beneath and a complete dislocation result. Richet has verified a case of this kind at autopsy; the face was rotated to the right, the chin almost resting on the clavicle of the right side. Above the site of injury the spine was convex posteriorly. If a break in the continuity of the line formed by the transverse processes can be demonstrated, it is pathognomonic of the condition. Examination through the mouth can further show that the vertebræ situated above the site of dislocation are *anterior*, and, at the same time placed *transversely* to those situated below. It is not surprising that pressure symptoms, due to injury of the cord, occur.

Important from the standpoint of differential diagnosis are the cases in which the dislocated vertebra has been impaled upon the superior articular process of the subjacent vertebra—a so-called *incomplete* unilateral dislocation. I saw a case of this kind with Schuh, and another, as a student, with v. Dumreicher.

The cook of a well-known physiologist met with this accident one morning by suddenly turning her head while still in bed. The head was found flexed and turned toward the right; the right side of the cervical spine concave, the left convex; neck and head both completely fixed. At the apex of the convexity, on the left side, was a painful spot along the transverse processes. *The muscles along the concave side were not contracted;* radiating pain was felt along the brachial plexus. Schuh undertook to reduce the deformity and succeeded at the first attempt.

Other peripheral symptoms may be present; for instance, vasomotor symptoms, such as bright flushing of one side of the face, which is also warmer.

This case of Schuh's, which possesses all the points needed in diagnosis, may profitably be compared with the following case seen by Dupuytren:

> A fifteen-year-old boy, engaged in changing his linen, made a sudden movement, which was immediately followed by a violent pain in the nape of the neck. At the same instant the patient plainly heard a cracking sound. His head bent over to the left and remained fixed in that position. The physicians, who saw him, sent him to Dupuytren with the diagnosis of an incomplete dislocation. The head was found strongly bent toward the left, the spinous processes of the higher vertebræ unduly prominent, the neck arched out on the opposite side. At the vertex of the arc the patient suffered intense pain, increased by attempts to raise the head. He also complained of deafness, of shooting pain in the right arm and shoulder, dysphagia, and inability to rotate or flex the head. The majority of the physicians and students present concurred in the diagnosis of dislocation; not so Dupuytren. He pronounced it muscular rheumatism, and prescribed wet cupping along the left side of the neck. The correctness of this view was established next day by the fact that all the symptoms seen on the preceding day had disappeared so completely that the above description would have appeared incredible had it not been verified by all those present. The cervical spine had completely regained its mobility.

But how did Dupuytren justify his diagnosis? In his Leçons orales, from which the case is taken, his conclusions are based upon peculiar grounds. Dupuytren says: " I suspected rheumatism in this case because the boy's occupation, assistant in a liquor business, obliged him to spend many hours daily in a damp cellar, and also because he had previously suffered from rheumatic attacks." Although Dupuytren is well known for his logical deductions, he has failed in this instance to state his method of reasoning with sufficient clearness.

That the patient's symptoms did not harmonize with the assumed dislocation, Dupuytren saw at once; he therefore excluded dislocation. On the other hand, he had positive signs in favour of rheumatism. In what way did the symptoms differ from those usually seen in dislocation? Possibly in the amount of malposition—a purely quantitative symptom, and therefore of no value. Possibly the picture failed to agree with a sign previously mentioned in connection with Schuh's case—"the muscles along the concave side were not contracted." Perhaps, in Dupuytren's case, the muscles along the concave side were contracted, and yet the great clinician failed to emphasize this point. *In all cases of incomplete unilateral dislocation the muscles along the concave side of the neck are relaxed;* this is conclusive. If they are tense, dislocation may be excluded. One other characteristic should be kept in mind: In cases of dislocation, limitation of motion persists under anæsthesia until the dislocation has been reduced; in rheumatism, under these conditions, movement is free. The muscles may not be sufficiently contracted in all cases of rheumatism to permit of a positive diagnosis. The local point of tenderness should then be sought for. In dislocation this point will be found at the apex of the convexity; in rheumatism, the muscles of the concave side are tender and painful.

As rheumatic pains and stiffness of the neck frequently appear immediately after some sudden movement, the differentiation of dislocation from rheumatism is more often necessary than if rheumatic manifestations were slower in their onset.

The rheumatic pains known as lumbago are called *Hexenschuss* (witches' stitch) in colloquial German. The pain, and also the stiffness, begin suddenly. Dupuytren noticed—and experience constantly confirms it—that lumbago frequently first makes its appearance when the individual is stepping into a wagon. The victim turns in surprise to see who has wounded his back. The mode of onset of rheumatism and of trauma, therefore, resemble each other closely. Would that we knew what rheumatism really is! We have used the word for many centuries without definite conception of its nature. It is only in the last few years that the infectious character of the affection has become more widely recognised, but as infectious diseases not infrequently appear after traumata, a traumatic history does not necessarily exclude rheumatism.

There is a marked contrast between the precise and satisfactory signs of dislocation of the cervical vertebræ and the indefinite symptoms produced by FRACTURE.

Abnormal mobility is rarely obtained, and then only by some lucky chance. In other instances the peculiar deformity which results can only be explained by assuming fracture of the bones. The sign common to all fractures—abnormal mobility—is not obtained except on the cadaver. In the living subject it is replaced by the stiffness of posture. At times the diagnosis of fracture can be made by the grave symptoms (which, however, do not present the picture of a dislocation) resulting from the application of an adequate force. Such are, for instance, crushing of the cord, which can be explained only by assuming a displacement of part of the spinal column.

A case of Strohmeyer's is instructive in reference to *Contusions.* Following a contusion, the head was thrown so far backward, and was so persistently held in this position, that it was by no means unjustifiable to think of a bilateral dislocation. A strongly pronounced spasm of the muscles at the *nape of the neck* aided in the differentiation, and within twenty-four hours motion was restored.

We are indebted to the same author for a case illustrating another variety of injury—namely, *rupture of the ligaments* connecting the fifth and sixth cervical vertebræ. Intra vitem, motions of the neck were greatly restricted in all directions. The cause was discovered at autopsy.

Fractures of the first cervical vertebra give rise to a striking phenomenon, which is illustrated by the following case:

A girl, who had received a heavy blow on the neck, was seen by Cooper. Following the injury, whenever the girl wanted to look upward she was obliged to grasp her head with both hands, and, by degrees, raise it toward the desired object. If the object was nearer the ground, she grasped her chin and lowered her head. If another child bumped into her, the concussion produced a disagreeable sensation. The patient would run to a table, or any other stationary object, in order to rest her head against it, and stand, supporting her chin, until the excitement due to the accident had subsided. The child lived for twelve months. At the post-mortem, Cline found a transverse fracture of the atlas, without displacement. Raising the head caused the odontoid process to leave its normal position, at the same time dragging the atlas with it.

Especially to be noted in this case was the fact that the child used its hands to bend the head forward. It felt too insecure to intrust its head to the muscles of the neck. The findings at the autopsy explain this. An analogous condition will be mentioned farther on.

This completes the discussion of those conditions in which the *suddenness of the onset* of the symptoms governed the whole picture—in which, therefore, a rheumatic or traumatic etiology could alone be suspected.

It is self-evident that a patient suffering from anthrax of the neck will hold the head rigidly. In cases of anthrax, as well as in those of acute swelling of the lymph glands of the neck, phlegmons, and similar conditions, the position of the head is not the basis from which we reason. On the contrary, it is merely one of the evident consequences of the disease which is visible on inspection.

Let us now consider a chronic case, in which the patient has carried his head stiffly for some time, avoided turning or flexion of the head, and never looked to the side without turning the whole body. We assume that the disease has begun insidiously, without apparent cause. How is the diagnosis then made?

Starting with anatomical considerations as a basis, we naturally think of some disease of the muscles, of the bones, or of the joints. Or we can narrow down our scheme to two considerations: to that of a disease of the organs which produce motion and of those which are passively moved. In point of fact, this simplification is more in harmony with the clinical requirements. For the frequent and dangerous disease of the passively moved organs, which is next to be considered (*Pott's disease*), starts either in the vertebral joints or in the bodies of the vertebræ. Physicians of the old school are still in the habit of classing this as a caries, ostitis, or periostitis of the cervical spine. We know that, anatomically, the same tubercular process covers one or all of these names. Pott's disease, caries of the vertebræ, etc., are therefore more properly known as *Tuberculosis* of the spine.

In addition to immobility of the head, other symptoms are necessary to confirm the diagnosis of Pott's disease, these symptoms varying according to the stage of the disease. The following applies to typical cases:

1. At least one spinous process is sensitive, and, later on, unduly prominent — a very important symptom,

which at the same time is an example of how a superficial sign may point to a deep-seated process. As Pott's disease most frequently begins along the anterior surface of the bodies of vertebræ, under the anterior longitudinal ligament, the inflammatory destruction of the bone progresses more rapidly at this site. The weight of the head presses most heavily on the anterior part of the bone, which gradually assumes a wedge-shaped form, narrow in front, wider behind. The vertebræ immediately above and below necessarily rest upon two planes converging anteriorly, and the spinous process of the diseased vertebra must become unduly prominent. If the body of the diseased vertebra can be reached through the mouth, it will be found painful on pressure.

Distinct prominence of a spinous process is noticed only in fairly advanced cases, in which the vertebra is decidedly wedge-shaped—that is, considerably eroded anteriorly. In the early stages there exists only a lessening of the concavity of the neck (lessening of the physiological lordosis of the cervical segment).

The cervical spine is found almost straight, which virtually represents a curvature with its convexity directed posteriorly (kyphosis).

2. The further course of these cases may lead to the formation of retropharyngeal abscesses. These produce difficulty in breathing and swallowing, and can be readily palpated as elastic swellings in the posterior pharyngeal wall.

3. Symptoms referable to more distant parts lend further support to the diagnosis. The inflammation in the vicinity of the intervertebral foramina causes disturbances along the distribution of the spinal nerves,

due partly to mechanical pressure, partly to the extension of the inflammation along the nerves themselves.

Experience has shown that the disturbances are rarely motor. As a rule, they are neuralgic pains: occipital or cervical neuralgias, if the disease is located in the upper cervical region; brachial neuralgias, if in the lower. If the tubercular process is situated in the lateral portion of the vertebra (the articular processes or intervertebral joints) the picture does not conform to this type.

(*a*) On one side of the neck an enlargement of the bony parts is perceptible; the bone is also painful on pressure. This apparent thickening of the bone, noticed on palpation, is largely due to a swelling of the soft parts attached to the bony structures.

(*b*) Or the infiltrated portion of the cervical spine is at the same time longer, so that the diseased side must become *convex*, the healthy, concave. The cervical part of the column has a lateral, bow-shaped curvature, and the head is bent to one side. If the process has already advanced to the stage of destruction the opposite condition may result. The eroded portion becomes shorter, the cervical spine is *concave* toward the affected side, and the head is rigidly inclined toward the diseased side. It is always on the diseased side that changes in the bone are demonstrable; in the first case, thickening and pain, in the second, destruction and the symptoms of abscess. These abscesses, which in unilateral cases form to either side of the median line, point either in the lateral region of the neck above the clavicle or appear in the axillary region, after burrowing along the large fascial opening through which the axillary vessels and nerves pass into the axilla.

(*c*) In other cases rotation of the spine takes place. The painful and enlarged part becomes somewhat prominent anteriorly, and in some cases we can feel, by palpation through the mouth, that the bodies of the vertebræ are more prominent on one side. Occasionally a small, steplike projection can in these cases be felt between the bodies of two vertebræ. The seat of the tuberculosis is then most probably in the intervertebral joints.

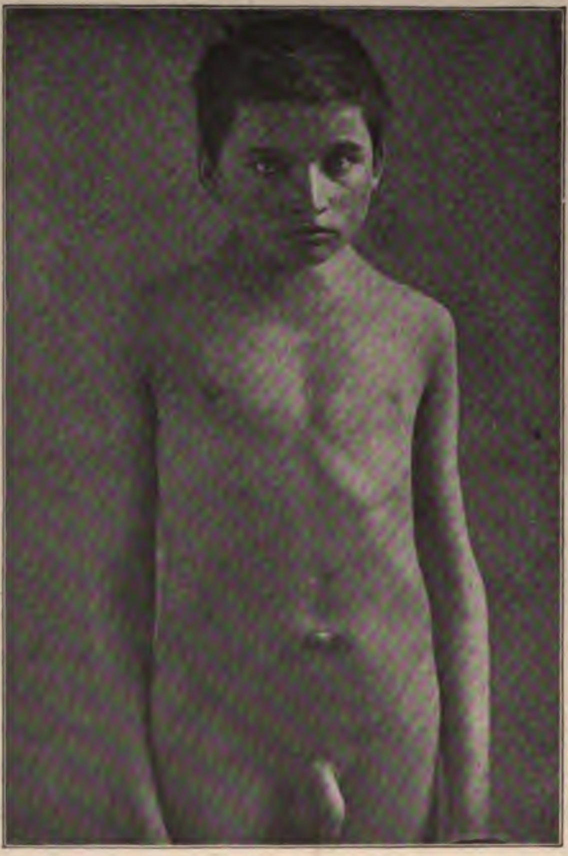

FIG. 1.

Both in tuberculosis of the bodies and of the joints of the vertebræ the rigid carriage of the head is the dominant symptom. We may suspect the disease as soon as the patient enters the door. He turns his trunk when he desires to look to the side, holding the neck stiff and immobile (see Figs. 1 and 2).

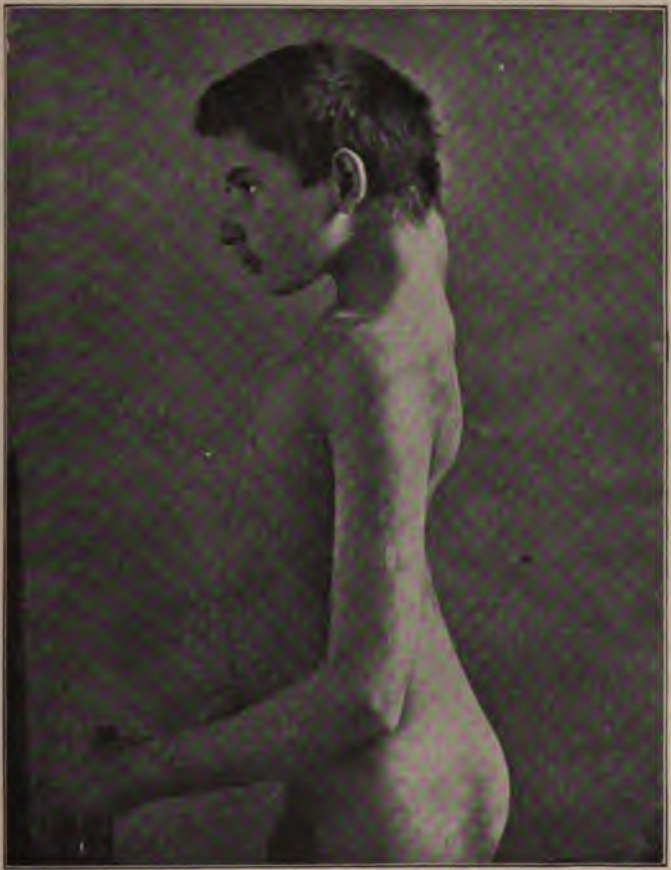

FIG. 2.

In that disease which Leyden has proposed to call *Rust's disease*, a peculiar position of the head is noticed.

This disease is merely a special kind of Pott's, a caries of the *uppermost cervical vertebræ*, more particularly a caries which occurs in the joint between atlas and the odontoid process. It well merits a special name and description, not only because carious erosion of the vertebræ in the neighbourhood of the vital centres of the spinal cord cause the most serious dangers, but also because the symptoms of the disease are highly interesting, and permit of the probable diagnosis with considerable certainty. As turning movements of the head take place in a horizontal plane about the vertical odontoid process of the axis, the head and atlas moving as one, it is evident that as a result of the inflammation of this joint turning movements of the head are entirely inhibited. Nodding movements take place in the joint between atlas and the occipital bone, and these are only partially or not at all impeded. Loss of the power of turning the head, with persistence of nodding movements, are, therefore, a diagnostic guide. The patient himself furnishes a further clew by supporting his head with *his hands* on changing from the sitting to the recumbent posture, and vice versa.

Some patients even lift the head by the hair when they attempt to rise. In some unknown way they divine, or unconsciously avoid, the danger which a sudden movement might occasion. In the end most of them meet a sudden doom through dislocation or fracture of the odontoid process and consequent crushing of the cord.

A further hint of great value in making a prognosis, if the other symptoms lead us to suspect the trouble, is furnished by the absence of all external signs. This points to the deep location of the disease.

Leyden has shown that the same symptom-complex is present when the odontoid process is the seat of *Carcinoma* or *Sarcoma*, with the exception that in malignant disease the pain does not abate when the parts are at rest. This disease is found in adults, and is very rare. It has been previously stated that in case of fracture the head is likewise fixed by means of the hands during all sudden movements.

With this the discussion of the bone diseases which cause abnormal positions of the head is completed.

Turning now to the muscular apparatus, we find at least *one* symptom to rely upon. The contracture of a muscle will not cause *immobility* of the head and of the cervical column, but merely a definite *limitation of movement* in some one direction. In contracture of the sterno-mastoid, for example, we meet with distinct resistance to those movements which put the muscle upon the stretch. In other directions movement will remain normal. This is the basis of our reasoning in determining whether the obstacle is situated in muscle or in other structures.

Of all the muscular diseases which cause a pathological position of the head, the contracture of the sterno-mastoid is the most frequent and most interesting. The condition is known as *Caput obstipum*, or *Torticollis*. In unilateral contracture of the sterno-mastoid the ear of the diseased side approaches the shoulder, and, at the same time, the chin is turned toward the sound side. Besides this, the occiput is bent slightly backward. If the presence of the other symptoms of this disease are taken for granted, it is necessary to determine from the standpoint of differential diagnosis whether, in any given case, the contracture of the muscle is primary or secondary. A cured Pott's disease can cause a similar posture due to a scoliotic distortion of the cervical column. In these cases the sterno-mastoid likewise stands out as a firm, cordlike structure, because its points of insertion have for a long time been approximated, and a real contracture or shortening has resulted. In some of these cases a single glance will suffice to make the correct diagnosis. If, with a rigidly prominent sterno-mastoid, the head is bent toward the same side, but the chin *not* turned toward the opposite one, we immediately suspect that the case is not one of primary contracture. This suspicion should be verified by examining the spine for the presence of deformity and for the degree of limitation of movement, in order to determine the presence of some healed bone lesion. If the contracture is primary, all movements, in which the sterno-mastoid is not put upon the stretch, are unimpeded. This wry-neck is purely *muscular*, and almost without exception congenital in origin, due either, ante partum, to some strained position of the head of the fœtus in utero, or to intra partum tearing or inflammation of the muscle.

In cases of contracture of this muscle occurring in adults, another point of doubt may arise. We may suspect some disease of the cervical spine; for there are cases of *spondylitis deformans*, analogous to malum coxæ, in which distortion of the neck is the final outcome. Here there is pain in the joints of the cervical portion of the spine, increase in size of the articular processes, and, not infrequently, cracking sounds on motion. Do not let an *hysterical* woman mislead you, for in this disease cramps and contractures of the sterno-mastoid occur, and during examination of the cervical column the patients complain of pain. In such cases, however, there is a disproportion between the high degree of contracture and the slight and varying amount of pain. The vertebral column is not at all deformed, and other signs of hysteria are present.

I was called to examine an eleven-year-old girl in whom one physician diagnosed Pott's disease, and another muscular spasm. The head was bent so strongly toward the right that the cheek rested upon the shoulder. Examination of the neck showed the spinous processes of some of the vertebræ slightly sensitive to pressure, but nothing else was to be found. It was impossible to move the head from its abnormal position, the patient complaining of intense pain at each attempt. Consequently I was unable to examine the muscles of the affected side for spasm. The physician who supported the diagnosis of muscular spasm gave as his reasons that the girl was strongly built, not anæmic, and free from hereditary taint. The girl really was well-built and muscular. He further stated that this high grade of contracture had developed in the course of a very short time; that no deformity or destructive process was demonstrable in the spine, and that the painful points changed their location. I was obliged to agree with these findings. After several months, the spasm disappeared. Later it was discovered that the father of the girl—an unusually neurotic subject—had suffered from the same trouble in his youth.

There are other neuropathic contractures of the neck muscles. The site of these contractures is indicated by the tense state of the contracted muscle. Contracture of the *trapezius* causes the head to turn toward the diseased side and to bend backward; the shoulder is somewhat elevated and pulled inward. In tonic spasm of the *splenius capitis*, the head is likewise bent toward the affected side, inclined backward, and the chin approached to the shoulder of the same side. Bilateral spasm of the sterno-mastoids causes the head to sink down upon the breast; spasm of the deep muscles of the neck bends the head backward.

The following example will show of what importance recognition of the significance of the position of the head may prove to be:

I was substituting for my teacher, v. Dumreicher, in Vienna, when I was informed that a child, urgently requiring tracheotomy, was about to be carried in. I immediately interrupted the lecture, and the child was introduced. Its breathing really indicated a high degree of stenosis of the air-passages. The most natural thing to think of was croup. But the child did not show the retraction of the head usually seen in croup. On the contrary, the head was bent forward and kept in this position without the slightest movement. This circumstance, combined with the pale looks of the child, caused me to suspect, at the first glance, that the dyspnœa might be due to caries of the cervical spine, with retropharyngeal abscess. Without delay I palpated the neck, and discovered a prominent spinous process. I now passed the fingers of my left hand into the child's mouth, and found an elastic bulging of the posterior wall of the pharynx. Introducing a knife, I opened an abscess, the whole incident occupying only a few seconds. The dyspnœa rapidly diminished, and I now had leisure to explain my actions to the students.

CHAPTER II

INJURIES TO THE SKULL AND BRAIN, AND THEIR PRIMARY SEQUELÆ

THAT the final outcome of injuries of the skull can never be predicted with any certainty has been known from the earliest times, and is a fact which still holds good. Severe penetrating wounds, complicated by crushing of large areas of brain substance, may heal without leaving any after-effects or any disturbance of the bodily functions. On the other hand, a blow on the head, which does not cause the victim to fall to the ground but which produces a small wound of the soft parts, may rapidly lead to erysipelas, meningitis, and death. In recent times antisepsis has proved a blessing in these cases, when applied at the right time, by preventing the appearance of suppuration. I have made these preliminary remarks in order to indicate in advance that in wounds of the skull—just as in wounds of the thorax and abdomen—antiseptic measures must precede all attempts at arriving at a more accurate diagnosis.

Injuries to the skull may be divided into:

1. Injuries of the soft parts.
2. Injuries of the bones of the cranium.
3. Injuries of the intracranial contents (brain, meninges, blood-vessels, nerve-trunks).

These injuries may be subcutaneous or open; the latter are known as wounds.

Accordingly, we distinguish wounds of the soft parts covering the skull, of the skull bones, and of the brain and its meninges. According to the weapon inflicting the injury, the wounds are divided into punctured, incised, tearing, lacerated, gunshot, etc. Such injuries may be accompanied by considerable loss of substance, the most striking example being the "lopping off" of a part known among the ancients as *Αποσκεπαρνισμος*. In cavalry encounters, a sharp sword, or a Damascus blade, occasionally lops off part of the skull; for instance, if the blow strikes the parietal eminence, a flap, consisting of soft parts, bone, and brain, may be entirely severed.

Subcutaneous injuries are represented by bruises of the soft parts, hæmatomata, fractures of the skull, bruises and rupture of the brain-matter, rupture of the internal blood-vessels (meningeal or cerebral hemorrhage), and of the nerve-trunks situated within the skull. To these we may add two peculiar varieties of injury—*compression* and *concussion* of the brain.

The whole attention of the physician who is treating an injury of the skull is first directed to determine whether the brain and its membranes have escaped. If the wound is compound, direct examination by sight, and eventually by the probe and finger, are undertaken as a matter of routine. If no wound is present, if the injury is subcutaneous, the involvement of the brain can be diagnosed only by means of certain functional disturbances.

It is best, therefore, to begin at once with one of these difficult cases, and to discuss by what signs an injury to the brain can be recognised.

Bruises, tears, pulpification, or breaks in continuity are always *local* lesions, no matter whether simple, and then due to the splinters of a depressed fracture, etc., or compound, as the result of an open wound. They affect only a circumscribed portion of the organ, the cortex, as a rule, being the part involved.

In compression and concussion the whole brain takes part. These two forms represent *diffuse* lesions.

As the brain functions are localized in discrete, circumscribed parts of the whole brain, theoretically, at least, we may hope to recognise the destruction of any part by the disturbance of those functions which are regulated by it. Unfortunately, however, our knowledge of the separate centres is yet imperfect. Consequently, LOCAL LESIONS OF THE BRAIN can be diagnosed only in a limited number of cases by means of the resulting disturbances of function.

A few instances will not be out of place. If a soldier receives a bullet wound in the side of his head, and then grows hemiplegic on the opposite side of the body, a destruction of the cortical motor areas may be diagnosed. If the patient, after trauma, suffers from aphasia, an injury to the left hemisphere, embracing the part known as Broca's speech centre, may be assumed to exist. In paralysis of the facial nerve, its centre has been injured, unless the position of the wound indicates that the trunk, and not the centre, has suffered.

Paralysis, however, is not the only evidence of a local lesion, though it shows that a part of the central nervous system has been destroyed, or has been rendered inactive. Some portions of the central nervous system can be injured or acted upon in such a fashion that they respond to this stimulus by irritative symptoms. A splinter of bone can penetrate and destroy one centre, but at the same time merely irritate an adjoining centre. The reaction following an injury can also produce symptoms of irritation in the neighbouring parts.

The deductions drawn from these facts are, that circumscribed paralysis, or circumscribed symptoms of irritation, but especially a combination of the two, point to a *local* lesion.

Circumscribed paralysis—i. e., paralysis limited to one group of muscles—is known as *Monoplegia*; circumscribed spasms are called *Monospasms.* If the spasms spread from the primarily affected group to other groups of muscles, the spasm which originated the phenomenon is known as the *Protospasm.*

The following combinations may be taken as types representing various local brain lesions:

Aphasia, with facial paralysis and paralysis of the upper extremity; proto- and monospasms of the facial; hemiplegia; hemiplegia, with spasm of the affected limbs; aphasia, with protospasm of the upper extremity followed by hemiplegia.

These various combinations can be explained by bearing in mind that the centres governing the extremities are contiguous, and that the centre for the facial and the centre for speech are situated in close proximity.

Spasms are the result of irritation, and this irritation soon leads to exhaustion of the centre; or a secondary inflammation may destroy the centre, which at first was merely irritated. This explains why paralysis of the affected group of muscles may follow protospasms.

Of the diffuse brain disturbances, CONCUSSION is by far the most frequent. As the name indicates, concussion represents the rapid molecular displacement of the brain substance. The causes are a blow or fall on the head.

If we examine a case of concussion of medium severity, the following striking combination of symptoms are noticed:

1. Immediately after the trauma the patient falls to the ground and lies *unconscious* for a period varying from five to fifteen minutes. He has no recollection of the accident (amnesia).

2. Immediately, or on recovering consciousness, *vomiting* sets in, and may continue at intervals for several hours.

3. The pulse is *slowed*, the rate per minute being 60, 56, or 48 beats, or thereabouts.

In these cases respiration also is somewhat slower, the surface temperature subnormal. When the patient wakes up he is but partly conscious, and remains somnolent, torpid, and without speech or motion.

Minor degrees of concussion evince themselves by a *dazed* or *stunned* condition following the injury. Hearing, sight, and cerebration are inhibited for the time being. The patient falls to the ground, but awakes in a short time from his unconscious condition. For several hours slight dizziness, tinnitus aurium, muscular weakness, and headache remain. Recovery takes place within one or two days without treatment.

High degrees of concussion of the brain present a very grave picture. Unconsciousness may last for hours, the patient lying as if dead. The physician finds him still comatose, with muscles wholly paralyzed and not responding to stimuli; or the condition may be one of stupor, with but momentary returns to consciousness. The body is pale and cool, respiration weak and superficial, the pulse small, irregular, and usually slow. Reaction, as a rule, follows, the body becoming warm, respiration deeper, pulse fuller, and consciousness and ability to move return. Instead of the pallor noticed in the first stage, the patient now appears flushed. One symptom may remain prominent for several days. This is the *slowing of the pulse-rate,* which may drop to 40 a minute, and yet the pulse continue to be full and of high tension. When the pulse returns to the normal rate the remaining symptoms—especially the headache, tinnitus, drowsiness, and slowness of speech—disappear.

The characteristic symptoms of concussion of the brain are evidently a *transient condition of depression.* If irritative symptoms arise, or the depression grows more profound, the condition no longer is one of uncomplicated concussion. Some other brain lesion then exists.

One point requires emphasis. Loss of consciousness, no matter how transitory, must have been present if the condition is to be regarded as concussion. This, therefore, is the most distinctive sign, while slowing of the pulse is the objective symptom.

As a rule, fracture of the skull is accompanied by concussion of the brain, for a force of sufficient violence to fracture the skull will suffice to cause concussion. There are cases, however, in which the force is concentrated upon so small an area of the surface of the skull that the bone is broken, but the brain escapes general concussion. This point is of importance from a medico-legal standpoint.

Compression of the brain (not to be confused with the normal brain pressure, which is constantly present) is caused by some new factor which increases the intracranial contents. The simplest example of this is an intracranial hemorrhage,as, for instance, rupture of the middle meningeal artery. The escaping blood is added to the already present cranial contents (brain and cerebro-spinal fluid). As the cranial cavity is non-elastic, the brain is compressed by the superadded blood. A similar example is furnished by a circumscribed depressed fracture of the skull. Here the intracranial contents is not increased by a new factor, but the cranial capacity is diminished, and becomes too small for its normal contents. Relatively the two cases are the same.

As a result of compression the following symptoms arise: Nystagmus, vomiting, circulatory disturbance (slowing of the pulse-rate), disturbance of respiration, general convulsions, coma, and death.

These, however, are the symptoms experimentally obtained on animals.

The picture at the sick-bed may follow one of two courses:

By compressing the tumour in an individual suffering from hernia cerebri, and thus increasing the contents of the cranium and raising intracranial pressure, the following symptoms result: *At times*, besides a few convulsive movements, the patient sinks into stupor and the pulse-rate is very much decreased.

After rupture of the middle meningeal artery, the patient first suffers from paralysis of the extremities of the opposite side. The paralysis then becomes general, consciousness is lost, and the pulse is slowed.

Both these clinical pictures have in common the loss of voluntary motion and of consciousness, combined with a slowing of the pulse-rate.

The theory of compression is still unsettled and under discussion. I do not desire to discuss a doctrine which is still doubtful or incomplete. I shall therefore content myself with this outline of the clinical picture, which can be used as a foundation for the theory of compression of the brain. The second form alone can, in the strict sense of the word, be called compression. If a large clot of blood rests upon a hemisphere, and if the brain surface is not only flattened but depressed, so that a true concavity exists at the spot at which the extravasation is situated, no doubt can be entertained but that the brain is *compressed.* It is not only pressed upon, but also depressed. True, at first the compression is merely local, but it has been experimentally ascertained that if pressure is applied to circumscribed portions of the brain, the increase of intracranial pressure is transmitted in all directions through the brain substance. The pressure, therefore, affects the whole brain, as the local pressure soon becomes general. The theory explaining this last fact has not been fully elaborated.

If a theory is not quite ready for us we may as well stick to old traditions. Now, what are these?

If consciousness, lost immediately after an injury to the skull, returns, we heave a sigh of relief and call the disease concussion. But if unconsciousness again sets in after a short interval, we try to explain this by assuming that an extravasation of blood has taken place in the meantime, and has now reached sufficient proportions to compress the brain. If, after the trauma, consciousness does not return—although the patient continues to live day after day—and if depression of the skull is present, we explain the case by declaring that the brain is compressed by the depressed fragment of bone. But if consciousness finally returns and the paralyses are recovered from, we assume that the brain has accommodated itself to the increased pressure, or that an extravasation of blood which was present has been absorbed.

There are cases in which the paralysis persists although consciousness returns. If the paralysis disappears, after elevating the depressed bone, the palsy was due to purely local causes; if it continues, it was due to contusion of the brain.

If we attempt to apply these theoretical conclusions to the clinical facts, we frequently come face to face with great difficulties. For, in the majority of cases, local lesions and compression of the brain are found in combination; it could hardly be otherwise. Primary symptoms of compression can result from only two causes; either from a depressed piece of bone or from an exudate of some size, often further complicated by a local lesion, for, in this latter instance, the force causing the injury frequently produces some lesion of the brain substance in addition.

As a rule, however, the problem is soon solved. In a large majority of cases the course of events is as follows: The diffuse lesion—that is, the concussion—is the first symptom. As this is recovered from, the local lesion, with its symptom-complex, comes into prominence. The injured person suffers from unconsciousness, vomiting, and slowing of the pulse. But, sooner or later, consciousness returns, and consequently compression is no longer present. Then, with return of consciousness, aphasia, hemiplegia, or monoplegia develop. Therefore, we are dealing with a local lesion.

A single difficulty still requires explanation. Some symptoms of a local lesion—for instance, paralysis in the distribution of a cranial nerve—can be present without injury to the brain itself. If a fracture of the base tears or compresses a cranial nerve, the functions of the nerve will be interfered with. Thus blindness, deafness, anosmia, paralysis of the facial or of the abducens, etc., may result. The same symptoms may be produced by a bullet or stab wound severing any of these nerves in their course. It is often impossible to decide, on the spur of the moment, whether the penetrating instrument has reached the inside of the skull or not. At such times we are left in doubt as to whether a nerve or a centre has been injured until the combination of symptoms clears up the uncertainty.

Let us take the case of a penetrating body—for instance, of a projectile—which produces a circumscribed paralysis in the distribution of a cranial nerve. Certain facts taken for granted, two possibilities are then to be considered. Either some portion of the brain, or the cranial nerve itself, may have been injured. If the bullet has entered the forehead, either the optic nerve or the optic centre may have suffered. A bullet penetrating in the region of the ear may injure the auditory or facial nerve, or may reach their centres. In such cases the question arises, Is the paralysis of centric or peripheral origin? Neurologists have made strenuous efforts to solve this question, which is of such vital importance in the diagnosis of nervous diseases. They have endeavoured to discover certain functional symptoms by which we might hope to distinguish peripheral from central paralyses. When, for instance, not all the muscles supplied by the facial are paralyzed (the orbicularis and frontalis retaining its function), and when the paralyzed muscles retain their proper electrical reaction, neurologists conclude that the lesion is central. However, these theories have not as yet been sufficiently developed to prove of much assistance to the surgeon. Familiarity with the course of the nerves still remains of prime importance. For instance, as the result of an injury, the function of two or more nerves, which are close together in some part of their course along the base of the skull, may be disturbed. Their centres in the brain, however, are known to be separated by a large interval. We therefore can conclude that the point of injury is situated at that spot in their course at which they are in close proximity. If disturbance of the function of *spinal* nerves (as hemiplegia) is added to disturbances of *cranial* nerves, the obvious conclusion must always be that the brain itself has been injured, for the tracts from the higher centres pass through the brain on their way to the spinal cord.

The following examples will be used to illustrate some of the rules mentioned above:

A man, fifty years of age, was picked up unconscious in the street and taken to the hospital. A lacerated wound, extending down to the periosteum, was found over the parietal bone. The patient's beard and clothing were soiled with vomitus. The pulse was about seventy-two; the face pale. When loudly spoken to he mumbled unintelligible words. As the vomitus smelt strongly of alcohol, and the patient's breath smelt of the same, the question arose whether the unconsciousness and vomiting were symptoms of alcoholism or of concussion. The pallor might have been accounted for by the coolness of the autumn night. During the dressing of the wound, which caused some pain, the profane and filthy protests uttered showed that the patient was suffering from the less dangerous of the two conditions. This was made still more certain when, an hour later, he lay in bed with flushed face, snoring peacefully, and with a pulse which had risen to eighty-four beats a minute.

Alcoholics suffering from concussion present a different picture. Most noticeable is the slowing of the pulse. Respiration is not snoring, more commonly superficial and inaudible, the face remains pale for a longer period, the facial expression is plaintive, and, as a rule, no answer can be elicited. Moans or sighs indicate the more serious nature of the injury.

A man who had fallen to the ground from a high ladder was brought to the clinic. The patient had remained unconscious for some time, but after he arrived at the hospital he gave his name and an account of the accident. He vomited several times in our presence. The pulse was only forty-six beats a minute. It was noticed that the patient bled from the right ear, and also from the nose. The bleeding from the ear was quite profuse. Both lids of the right eye were suffused with blood; there was no subconjunctival hemorrhage. The right side of the face was flattened and without its naso-labial fold, but the left retained the normal play of muscles. He was unable to close the right eye. On examination of the mouth, while the patient said "A" in a loud voice, the left half of the uvula was seen to wrinkle; the right did not.

We here had to deal first with concussion, for consciousness was lost, the pulse markedly slowed, and vomiting had occurred. In addition, a fracture of the base had also taken place. The bleeding from nose and ear, combined with paralysis of the right facial, permitted of no other interpretation. We were able to declare not only that the fracture extended through the right petrous bone, but also that it was situated at a level above the point where the great superficial petrosal nerve is given off, for the palate appeared paralyzed. The fracture may strike the facial at a lower part of its course and then reach the greater superficial petrosal nerve at some other point.

It was necessary in this case to find out whether the patient could hear with the affected ear. More careful examination showed that he was deaf on that side. Evidently the auditory nerve was compromised, and consequently auditory transmission interrupted. Therefore, the fracture passed through the internal auditory meatus.

If any subconjunctival hemorrhage, especially if actively progressive, had been present, we might have said that the fracture extended along the roof of the orbit. Hemorrhage from the bone would then have shown itself subconjunctivally.

A Fracture of the Base is present, beyond a shadow of doubt, if brain substance or cerebro-spinal fluid discharges from the nose or ear. Brain substance can be recognised as such by microscopical examination. If serum oozes from the ear, and we are in doubt whether it is cerebro-spinal fluid or merely blood serum expressed from a coagulum, or a serous fluid due to some ear disease, the course of the case will decide. Cerebro-spinal fluid—which is characterized by a low percentage of albumen and high percentage of sodium chloride—continues to run for days, and in large quantities. In a case reported by Kelly nearly 300 ounces were collected between the second and fourteenth days. After this the outflow ceased. From the outset certain signs which point to the nature of the fluid may be prominent. Cerebro-spinal fluid flows more rapidly when the head is inclined toward the injured drum membrane, or when the patient makes strong expiratory efforts.

If to the flow of cerebro-spinal fluid is added paralysis of a cranial nerve, we account for this paralysis by assuming a fracture of the base, especially if the affected nerve is either the facial or the auditory, which traverse the petrous bone. If bleeding occurs from ear, nose, or mouth, separately or together, and paralysis of a cranial nerve occurs at the same time, a fracture of the base *is highly probable.* This is especially true if the bleeding from the ear continues for any length of time. (Under the same conditions a fracture of the base is *fairly* probable even if the paralysis of a cranial nerve is not present.) If, however, signs of fracture of the base are wanting, though the signs of concussion and paralysis of a cranial nerve are to be seen, the possibility of a contusion of the brain is not excluded.

> A brick fell upon the right frontal region of a man thirty-five years of age. Unconsciousness for ten minutes, followed by headache and dizziness. In four days these symptoms disappeared, but after the accident the left arm could not be properly moved. On the fourth day involuntary twitchings suddenly appeared in this arm. . . .

The rest of the history need not be quoted, but from what has been stated it is evident that a very moderate degree of concussion had been sustained. As paralysis of the left arm followed the injury, some local lesion of the right hemisphere must have occurred. It is probable that this was situated in the upper part of both ascending frontal and parietal convolutions. The spasms noticed on the fourth day were the result of reactive processes at the site of injury.

Finally, one more illustration. I will use a case of Tillaux:

> A man, run over by a cab, was brought to the hospital without any skin wound, depressed fracture, or disturbance of general condition.

After six hours, paralysis, first of the left, next of the right extremities, supervened. This was followed by coma and death within three days. Autopsy showed an extravasation of blood between the left parietal bone and the dura. The blood-clot weighed 140 grammes and caused a flattening of the brain. The surface of the right hemisphere was bruised by contre-coup.

Let us analyze this case from the point of view of diagnosis. The symptoms of paralysis increased with such rapidity that but one cause could be assigned. This cause is arterial hemorrhage, which is always rapid. It has been proven that the middle meningeal artery can *rupture* even without injury to the cranial bones. This rupture may occur on the side opposite to the blow, merely as a result of the momentary change in the contour of the bones. A sinus may tear, with consequent hemorrhage, but the bleeding is never as extensive. Why did the paralytic symptoms not manifest themselves at once? This fact is characteristic of *injury to the middle meningeal artery.* The blood must first separate the dura from the bones before it can balloon out the membrane and thus exert pressure upon the brain. As soon as this is accomplished, pressure symptoms rapidly develop. But why did paralysis first appear in the *left* extremity when the *left* hemisphere was injured? With compression of the left hemisphere a right-sided hemiplegia was to be expected. Probably the contusion had previously impaired the circulation of the right hemisphere, and the pressure was therefore more readily felt in that region. Was the left hemiplegia not the result of the right-sided contusion of the brain? No; for if it had been, this hemiplegia would have appeared immediately after the injury.

These are the types most commonly seen in ordinary practice.

Let us now turn to the *local examination* of injuries to the skull. There is naturally a considerable difference between subcutaneous and compound injuries. The outcome of simple fractures of the skull resemble the result of similar injuries of the ribs or pelvis.

When the force has not fatally injured the inclosed organs the fracture heals with or without disturbance of certain functions. If the injury to the intracranial contents (or to the contents of thorax or pelvis) is of itself fatal, it is of no importance whether the fracture was simple or compound. We may therefore say that in the case of severe simple fractures of the skull there is good hope of recovery, while in case of even an insignificant compound fracture the danger of a fatal termination must never be lost sight of. If the internal injury is not fatal, the subsequent course depends primarily upon the asepsis observed. This is the A to Z of the whole matter. Therefore, every examination must be conducted with strictest asepsis or antisepsis.

What should be looked for in examining the wound? In practice this really reduces itself to finding out whether there is a circumscribed depression or not. If it is present, we trephine or elevate the fragments. A foreign body, if present, should be removed. During the whole examination only these two points need be borne in mind. In many cases the examination is part of the treatment.

If the splintering is circumscribed, we remove all the movable fragments and smooth the circumference of the wound. If the fracture is of considerable extent, we content ourselves with removing the loose splinters, but refrain from excessive elevation, resection, or extraction. If it is a fissure, pure and simple, an antiseptic dressing will suffice.

In case of subcutaneous injuries our local examination should be directed chiefly to determine the following points:

In the first place we must decide in what layer a hæmatoma is situated; in the second, provided the hæmatoma is deep-seated, whether the bone has been depressed or not.

The following signs are of value: A hæmatoma which is directly under the skin is prominent and tense, and moves readily with the skin. A subaponeurotic or subperiosteal hæmatoma is, first of all, greater in extent, because the aponeurosis of the occipito-frontalis does not yield to pressure. It is therefore flattened, under low tension, and firmly attached. We might ask how a subaponeurotic extravasation could be distinguished from a subperiosteal. By introducing a needle this question may be satisfactorily answered; for if the needle strike a rough surface, the bone has been laid bare and the extravasation is consequently subperiosteal.

An inexperienced man may be readily led astray by the second query, whether the bone below a hæmatoma has been depressed or not, owing to the fact that a deep, especially a subperiosteal, extravasation often, or regularly, is bounded by a harder wall. The examining finger discovers this ridge and sweeps over it; the finger then sinks into the hæmatoma, so that the false diagnosis of a depressed fracture of the bone may be made. The impression is uncanny, but after examining a few cases this feeling of uncertainty is lost. The ridge about the extravasation is caused by a bloody infiltration of the tissues at the edge of the hæmatoma. Consequently the finger can make an impression in the ridge, in some cases even flatten it completely here and there. A feeling of very delicate crepitation, due to the friction of the blood coagula in the tissue spaces, may be obtained. No hesitation need be felt about making this examination, as the patient suffers no harm by it.

If a sabre wound extends along a markedly curved segment of the skull, and if the wound is long, we may generally suspect that at least the centre of the wound communicates with the cranial cavity. This assumption has more or less of a geometrical basis.

If, in an incised wound of the scalp, the edges of the wound do not gape, the injury has been confined to the skin; if the edges separate, the galea has been severed. Anatomical facts explain this, for the aponeurosis is drawn upon by its muscles, and thus separated. As the skin is closely connected with it, the wound gapes.

If a hæmatoma, or swelling, appears at a point opposite to the point of entrance of a shot, the projectile may be situated under this spot. Larrey found a bullet by cutting down upon the suggillation.

If the unharmed dura, exposed by some defect in the skull bones, shows no brain pulsation, one of several conditions may be present. The part of the brain situated beneath this point may be anæmic, or contused; or there may be an extravasation of blood under the dura. The anæmia may be caused by a splinter of bone wedged in between the dura and the skull. Contusion interferes with pulsation, because the contused parts have their circulation disturbed through destruction of the vessels. A blood extravasation situated beneath the dura will impart a bluish colour to the affected area.

CHAPTER III

INFLAMMATORY COMPLICATIONS FOLLOWING INJURIES TO THE SKULL

In many cases, carefully executed antisepsis will determine whether a penetrating injury to the skull will be followed by suppurative inflammation of the intracranial contents. Frequently, however, reliable antisepsis is out of the question. For instance, in fractures of the base, which involve the tympanum or nasopharynx, and consequently tear the mucous membrane, the pyogenic cocci find a point of entrance in spite of all possible precautions.

The most frequent complications to be considered are meningitis, brain abscess, and phlebitis of the sinuses. Other causes in surgical practice may, however, occasion these diseases. Thus, tuberculosis of the petrous bone or a suppurative otitis media may readily lead to an abscess in the neighbouring parts of the brain, or to inflammation of the meninges. A phosphorus necrosis may spread to the sphenoid bone and cause a meningitis. Erysipelas of the scalp may travel along the emissary veins and give rise to purulent meningitis, often with sinus thrombosis. Analogous complications may arise from a furuncle of the face. Following a resection of a cranial nerve infection has been known to travel along the nerve-sheath and enter the skull. I even saw a case of double sinus thrombosis of the cavernous sinus with fatal meningitis, shown by autopsy to have been due to an infection which had as its starting-point a carious tooth.

Abscess of the Brain demands our greatest attention in the study of the three chief varieties of secondary inflammations found in the interior of the skull—meningitis, abscess, and sinus thrombosis—because opening the abscess is a life-saving operation. It is true that many false diagnoses are made, and many purposeless trephinings have been done; but in the last few years successful cases have considerably increased in number because diagnosis has become more certain. Brain abscesses due to a foreign body have most frequently been opened, but here the foreign body served as a guide to the surgeon. If the operator discovers the foreign body which has become wedged in the skull bone, and, upon removing it, finds pus flowing out of the wound, he deserves credit only for finding and removing the foreign body, or at the utmost for enlarging the wound in the bone.

We intend to discuss chiefly the chronic brain abscess, which anatomically is characterized by an inclosing pyogenic membrane, and which is embedded in the brain substance just as a cyst would be. In recognition of the pyogenic membrane it is known as an encysted abscess of the brain. It is most frequently due to traumata which have caused some foreign body to penetrate and remain fixed in the brain. Next in frequency it is due to suppurative foci in the petrous bone. Third in frequency these abscesses are the result of metastases, and with greatest relative frequency of metastasis caused by endocarditis or by a septic process in the lung. Operative interference is naturally confined to the first two varieties.

What are the symptoms of a brain abscess? By *a priori* reasoning we would, in the first place, expect the destruction of a part of the brain to be followed by circumscribed paralysis, just as is the case in the destruction caused by apoplexy, and this is quite true. Without the symptoms of some diseased focus in the brain (paralysis with or without convulsions) a positive diagnosis of brain abscess is impossible. But, unfortunately, softening of the brain following a contusion of the brain substance gives the same symptoms. Experience has also shown that abscesses of considerable size may exist for months, or even years, without the patients suffering the least disturbance of function. Only at the very last, when the abscess, which has been embedded in the brain substance, bursts into the ventricle or through the pia, terminal symptoms rapidly develop. In the second place we would anticipate *a priori* that suppuration in the brain would cause pressure symptoms through the tension existing in the focus; but this is, again, not the case.

The preceding remarks, showing, as they do, that patients with brain abscess may seem perfectly well, disprove it. It is true that a slight increase in pressure (hyperæmia) may cause pressure symptoms to appear without warning. These manifest themselves as dizziness, a tendency to vomiting, and eventually produce stupor, which may again disappear as the pressure declines.

A chronic or encysted abscess may be suspected by its course. An injury to the head, accompanied by slight primary cerebral symptoms, is succeeded by a period of undisturbed health which may last for months or years. This is then followed by intermittent or continuous periods of headache, which constitute a particularly valuable symptom if they originate at the site of the injury. The patient suffers from dizziness and occasional attacks of vomiting, especially upon making some sudden movement of the head. Convulsions occur periodically, and evening attacks of fever are the rule. The abscess may be diagnosed, even at this time, if pus discharges from a small opening in the skull close to where a foreign body entered. Most likely a foreign body or a necrotic splinter of bone can be removed, the discharge of pus follows, and the probe passes down into the depths. On enlarging the opening in the bone we come upon a small slit in the dura, which, when dilated, gives vent to a large quantity of pus.

Or severe complications arise: severe attacks of fever and convulsions of the most diverse kinds; the headache becomes terrible; the patient grows delirious, and paralyses are noticed. Especially when hemiplegia on the side opposite to the injury occurs, the probability of a brain abscess is great if the general course of the disease has followed the type above described. If, relying upon the diagnosis, the abscess is opened by trephining, all these symptoms may promptly disappear; but if the disease is allowed to run its own course, high fever and convulsions are followed by stupor, œdema of the lungs, and death.

An especially doubtful picture is presented by the so-called *pachymeningitis suppuratoria externa circumscripta.* Heinecke relates a case in which no extension of the inflammation to the sinuses and to the pia took place. A blow upon the head was followed by fever and headache. A swelling, which pointed and discharged pus, formed.

A small opening in the bone admitted into the interior of the skull. On enlarging the opening with a chisel an abscess cavity between dura and bone was reached. There is no doubt that this was a case of osteomyelitis and necrosis of the skull bone, which had been bathed by pus on both its surfaces. But if any symptoms of meningitis had existed at the time of opening, we would have expected to find an abscess of the brain; only the fact that the dura was uninjured made this diagnosis improbable. Occasionally an idiopathic infectious osteomyelitis of the bones of the skull occurs and runs an analogous course.

Brain abscesses, secondary to suppurative otitis media, result either from direct extension from the bone to the meninges, or through metastatic progression of the cocci into the deeper parts, with an apparently healthy layer of brain substance between the ear and the abscess. Such abscesses, following disease of the middle ear, are legitimate objects for surgical interference, and in more recent years many successful trephinings have been accomplished.

Clinical experience has shown that suppuration starting at the tegmen tympani or in the anterior cells of the mastoid process, causes abscess in the temporal lobe, while suppuration of the posterior wall of the middle ear, or of the posterior mastoid cells, causes abscess of the cerebellum.

Sinus Phlebitis is much more readily diagnosed than brain abscess. Even if the general picture resembles meningitis in some of its details, we have three means of differentiation:

In the first place the etiological factor is of assistance. If, for instance, we are able to see phlebitis of a superficial vein during the course of a suppuration in the face or scalp, and this is succeeded by symptoms of meningitis, we are justified in concluding that the inflammation has travelled along the natural channels until it has reached the sinuses. In the second place, certain anatomical relations between the sinuses, nerves, and veins produce definite symptoms which point to a particular sinus. In the third place, in sinus phlebitis certain symptoms not present in meningitis occur; such are atypical recurring chills, jaundice, metastatic pneumonia or pleurisy, suppuration in the joints, etc., all of which are symptoms of pyæmia. They are due to direct infection of the blood by way of the sinus.

There remain to be mentioned only those peripheral symptoms which permit the localization of a phlebitis to some particular sinus. As the oculomotor, the trochlear, and the first division of the fifth lie on the side of, and the abducens traverses, the cavernous sinus, inflammation of this blood channel causes the following symptoms referable to these nerves: Pain in the frontal and supra-orbital region, ptosis of the upper lid, strabismus, cloudy or sometimes softened cornea. As the veins of the eye pour their blood into the cavernous sinus, œdema of the lids and of the subcutaneous tissue results from the arrest of circulation; also exophthalmos and immobility of the eyeball. If these symptoms appear in the opposite eye, the process has extended from the sinus of the one side to that of the other. If the phlebitis occurs in the domain of the transverse sinus the inflammation may spread directly into the upper bulb of the jugular vein. The nerves which pass through the jugular foramen are then pressed upon and paralyzed by the exudate and the swelling of the surrounding parts. This may cause a marked increase in the rapidity of the pulse, or fluctuations of the pulse-rate, aphonia, inability to swallow or cough—in short, to marked phenomena in the distribution of the vagus, the spinal accessory and glosso-pharyngeal nerves. An additional manifestation of utmost importance is the occurrence of convulsions. If the inflammation spreads farther along the course of the internal jugular, the side of the neck becomes swollen, the external jugular prominent, and the swollen parts grow extremely tender and painful. Inflammation of the superior longitudinal sinus produces epistaxis and general epileptiform convulsions, with headache at the vertex. In some acute cases the progress of the disease from one sinus to another can be followed by means of these landmarks.

Sinus Thrombosis is distinctly different from sinus phlebitis. As, however, most cases of sinus thrombosis terminate in a sinus phlebitis as a result of the disintegration of the thrombus, it becomes a question of diagnostic interest whether the thrombosis can be recognised before the secondary inflammation has started. In isolated cases it can be suspected when the external symptoms of an arrest of the intracranial circulation occur, such as increased prominence of the eyeball with œdema of the lids and injection of the conjunctiva, the presence of unequally distended external jugular veins, etc. We may also suspect a thrombosis if cerebral symptoms are noticed—vomiting, headache, and delirium—which develop rapidly and then diminish, though they may recur with equal rapidity. Finally, in disease of non-inflammatory origin, such as marasmus (marantic thrombosis) or tumour, these symptoms will lead us to believe that the sinus has been compressed or its lumen encroached upon.

The symptoms of *Meningitis* due to trauma or to the extension of inflammation from neighbouring parts differ in no way from those of meningitis due to other causes.

For surgical reasons meningitis of the hemispheres is distinguished from that of the base, so that we speak of *meningitis of the convexity* and *basilar meningitis.* Even if the points of differentiation known to us are not absolutely trustworthy, their discussion is very suggestive, and may lead to further increase in our knowledge. The cases which come under the observation of the surgeon are clear, at least in one respect, as the local point of origin of the process is usually known (scalp wound, caries, or necrosis of the skull). This shows us, at least approximately, how the anatomical progress of the disease is marked by corresponding symptoms.

In all cases of meningitis we distinguish a stage of irritation and a stage of depression.

The stage of irritation is marked by psychical excitement, increase in pulse-rate and temperature, convulsions, contractions, hyperæsthesia, and headache.

In the *stage of depression* excitement is succeeded by unconsciousness; among the motor phenomena paralyses become the prominent symptom; even the reflexes are abolished. Micturition and defecation are involuntary; the fever persists, but the pulse is slowed, while respiration grows stertorous. Only toward the very end respiration again becomes rapid and the pulse more and more frequent.

It is necessary to bear in mind that symptoms of irritation (like convulsions) may be combined with symptoms of depression (such as paralysis), and further, that those groups of muscles which were at first irritated are not necessarily the ones which later grow paralyzed. The affair is not quite as schematic as this. Contractures, convulsions, and paralyses may occur together; for instance, ptosis of one lid, strabismus, and at the same time clonic spasms of an extremity. Groups of muscles which previously were not affected by convulsive twitchings may be paralyzed. The division into two stages is founded upon the general picture presented by the patient. In the first stage he is excited and delirious; in the second, comatose and stuporous.

Meningitis of the convexity is distinguished by the occurrence of hemiplegia, or at least of hemiparesis. This is explained by the assumption that the inflammation of the meninges renders the immediately subjacent motor cortex incapable of performing its functions. Bergmann has employed the following analogy: He believes that the process is similar to that seen in peritonitis, where an inflammation of the visceral peritoneum is followed by a paralysis of the underlying muscular coats. Basal meningitis runs its course without hemiplegia.

All in all, we must remember that meningitis is a disease which shows itself in a great variety of forms. As it may appear primarily (in the first few days following a trauma), or also secondarily (in the second or third week), after an injury to the skull, we must be on the watch for it, and give a very guarded prognosis.

If it is noticed that the injured person evinces a marked restlessness, that he tears the bandages from his head, grinds his teeth, spits, and, on being questioned, complains of headache, these symptoms must not be regarded lightly. If the temperature now rises more rapidly, our anxiety is increased. If vomiting sets in, the patient on the next day will show ptosis or facial paralysis, or the attendants report that the patient has

had convulsions. This shows that meningitis is already present.

In certain cases we may be deceived at the beginning of the period of irritation. For instance, following a case of fracture at the base, with severe concussion, the excitement of the patient may indicate the beginning of a primary meningitis, or perhaps no more than the period of excitement following upon a severe concussion. In the latter case, suitable treatment (rest, cold, purgation, or, if the pulse is hard and slow, venesection) will soon cause the disappearance of the symptoms.

The following case was seen by me at the beginning of my surgical career: During my period of service under Lorinser there was a woman about fifty years of age in the ward. She was suffering from tuberculosis and caries of the calcaneum. Suddenly, in the course of one night she became hemiplegic. Lorinser, making his rounds, saw her only in passing on three successive days, and merely stopped to feel her pulse and to have her show her tongue. On the third day the patient died. When asked what cause of death we should put on the death certificate, he answered, "Meningitis." We were surprised when, on autopsy, purulent meningitis was really found; and were eager to discover how Lorinser was able to diagnose the case with such superficial observation. The answer which we received has always remained fixed in my mind: "As the patient was not suffering from any circulatory disease, but from caries and tuberculosis, apoplexy was unlikely. The sudden onset of hemiplegia was indeed striking, but this continually increased in severity. For on the first day the patient's tongue was protruded to one side; on the second, much more so, and on the third, even more. This pointed to a slowly progressing process, in other words, to an inflammatory affection. On the third day the patient's pulse grew slower, in spite of the increase in the paralysis, and in spite of her having suffered for weeks with fever. If you had watched the patient more carefully you surely would have discovered other symptoms of meningitis." We really discovered, upon questioning the patient in the adjoining bed, that the deceased had suffered from convulsions during the night, which the nurse had failed to report.

CHAPTER IV

TUMOURS OF THE SKULL

A SERIES of very interesting tumours are found on the skull.

The foremost in importance is *Hernia Cerebri.* Just as in all other herniæ, we distinguish hernial orifice, hernial sac, and hernial contents.

The point of exit is formed by the non-approximation of two or more skull bones. At this spot a gap exists, and the name given to each hernia depends upon the bones which have failed to come together and have thus given rise to the hernial orifice, such as a naso-ethmoidal or spheno-orbital hernia, etc.

The *hernial sac* is formed by the dura mater.

The *hernial contents* varies in character.

(*a*) It may consist of a saclike protrusion of the arachnoid filled with cerebro-spinal fluid derived from the subarachnoid space—*Meningocele.*

(*b*) Of a part of the hemisphere, into which, however, the ventricle is prolonged—*Cenencephalocele.*

(*c*) A more involved hernia, in which, owing to disappearance of the brain substance, the ventricle opens directly into the hernial sac.

The coverings of a hernia cerebri are composed of the meninges. In herniæ of small size their aspect may be normal; in larger ones they are, as a rule, thinned out. The membranes may be thickened by œdema or by deposition of fat, or at times show peculiar folds resembling the skin around the navel.

Occasionally it happens that such a hernia is shut off from the cranial cavity, its point of exit closing behind it. If this occurs in a hernia situated in the sagittal suture, a cystic swelling is formed in the region of the large fontanel. Such a swelling is firmly fixed in a trough of bone. The hernia consists of a serous fluid (cerebro-spinal or arachnoidal fluid) surrounding a cheesy material (the altered brain substance).

Besides this, cysts *superficial* to the dura occur, which protrude, toward the surface, through a gap in the bones.

Next in interest are those *Blood Cysts* which communicate with the interior of the skull. They are either protrusions of the blood channels (as of the longitudinal sinus through the sagittal suture, of the lateral sinus in the occipital region), which are true analogues of hernia cerebri, or rupture of the diploic veins. In the latter case the swelling is analogous to a false traumatic aneurism, and consequently is known as a false traumatic varix (varix traumaticus spurius).

Next to be considered is the *Sarcoma of the Dura*, which penetrates the bones of the skull and then rapidly spreads in the soft parts composing the scalp. In its course it may cause ulceration of the skin, as it involves the parts over a large area.

These tumours, which form a group, have one point in common: they originate or have some connection with the interior of the skull.

As a matter of course, we next consider the tumours which take their point of origin from the bones of the skull. Exclusive of syphilomata and of those metastatic neoplasms which, according to Kundrat, are most commonly seen in connection with primary tumours of the parotid gland, this group is represented by *Sarcoma of the Diploe.* This sarcoma, which is quite rare, starts in the diploic space, and grows both inward and outward. If, in a given case, it has perforated the lamina vitrea, it may involve the dura mater, and is then readily mistaken for a sarcoma of the dura which has penetrated the skull.

Hæmatoma of the skull occupies a distinct place in this classification, for it is situated between the bone and the periosteum.

The tumours, which are situated in the scalp itself, form a third group. They consist of sebaceous cysts, cavernous angiomata, cirsoid aneurisms, papillomata, lipomata, etc., and offer no difficulty in their differential diagnosis. The only ones requiring differentiation are sebaceous cysts and dermoids. Dermoids are congenital, situated in the neighbourhood of the eye, especially about the margin of the orbit; they are more tense, usually more sessile, and embedded in a shallow trough of bone.

The *Cephalhæmatoma* of the new-born, on the whole, offers no difficulties in diagnosis. It is found in the newborn infant upon one of the parietal bones, and never oversteps the boundary of a suture. In form it may be circular, kidney, or pear-shaped. The swelling is distinctly elastic, and surrounded by a ridge. The edge may become bony if the periosteum, which has been raised by the effusion of blood, forms bony lamellæ. The bone formation may progress and involve the greater part of the swelling, so that palpation may give the sensation of parchment crackling. As, in rare instances, tuberculosis of the parietal bone with a subperiosteal abscess of some size is found, even in infants, this condition alone requires differentiation. But tubercular abscesses are invariably less tense and never show even the least indication of bone formation. In addition, they do not occur immediately after birth.

A difficult problem is furnished in the diagnosis of a sarcoma of the dura from sarcoma of the diploe.

Let us take, for example, a tumour attached to the skull, which, from its various characteristics, is evidently sarcomatous. The tumour pulsates. A sarcoma of the dura pulsates from the fact that the brain pulsations are transmitted to it. A sarcoma of the diploe may pulsate because of its vascularity. In the first place, we try to find out whether the tumour can be reduced. If this is the case, we most likely have to deal with a sarcoma of the dura which has been forced out by pressure through the broad gap in the bone produced by absorption. At the moment at which we succeed, evidences of pressure on the brain, or irritation of that organ, may occur.

Taking for granted that the tumour can not be reduced, a sarcoma of the dura is by no means excluded if the tumour be of sufficient size. We therefore carefully look for lamellæ of bone anywhere over the surface of the growth. The occurrence of these is a decided evidence in favour of its diploic origin; it is the remains of the outer table which was raised up by the advancing growth. Especially any bony cover felt at the edge of the tumour may be considered positive evidence of a sarcoma of the diploe, for a sarcoma of the dura never shows this characteristic.

Subjective symptoms speak more or less in favour of a sarcoma of the dura, especially headache, dizziness, and vomiting, occurring before the appearance of any visible tumour.

The group first mentioned, consisting of tumours directly communicating with the contents of the cranium, must now be considered in greater detail.

The following characteristics are shown by a blood cyst communicating with the cranial cavity—i. e., ectasis of a sinus (or a false traumatic aneurism), otherwise known as a *cephalhæmatocele*:

1. The bluish colour indicates a bloody contents. 2. Great and rapid variations in volume, depending on intrathoracic pressure. When, during expiration, the pressure in the thorax is considerably increased, the tumour rapidly swells up; when the intrathoracic pressure sinks, the blood is rapidly emptied from the swelling. 3. No brain symptoms on compressing the tumour.

The question of differentiating an ordinary varix of a vein of the scalp from a cephalhæmatocele now arises. The diagnosis is made as follows: A varix does not refill on coughing, screaming, or during deep and long-continued expiration after it has been emptied and its base is compressed. A cephalhæmatocele will refill in spite of circular compression of the surrounding parts, for its blood supply is not furnished by the compressible outer veins, but is derived either from the veins of the diploe or from the superior longitudinal sinus. If we have to deal with a false traumatic varix, the skull at the point of injury is uneven.

A typical hernia cerebri shows the following characteristics:

1. A distinctly cystic swelling (rounded, fluctuating, translucent).

2. The swelling shows the respiratory movements, and the pulsation of the brain, but the first never to the degree shown by a cephalhæmatocele.

3. It is situated along the meeting-place of one or more of the skull bones; a bony ring can be detected at its base.

4. It is congenital, or acquired after severe injuries to the skull (in which case it may be situated in the middle of a bone).

5. In some cases compression of the tumour produces symptoms of brain compression. In many cases, however, compressibility, and, consequently, pressure symptoms, can not be demonstrated. The diagnosis is then made by means of the situation of the tumour, the gap in the bone, and the fluctuating consistence.

CHAPTER V

THE FACE: TRIGEMINAL NEURALGIA

SPENCER WELLS, the most distinguished ovariotomist of our day, describes a characteristic facial expression in his work on Diseases of the Ovary. Among the profession it is usually known as " facies uterina "; he prefers to call it " facies ovariana." " The emaciation, the distinctly outlined muscles and bones, the expression of fear and of suffering, the furrowed brow, the deep-sunk eyes, the open, sharply outlined nostrils, the long, compressed lips, the drooping corners of the mouth, and the deep wrinkles which curve around it, together form a characteristic picture." This description, which accompanies Spencer Wells's illustration, is so general, that it will fail to produce in the reader's mind the deep impression created by observing one such patient. No matter how accurate this picture, no one will make a diagnosis of ovarian cyst, or even of any trouble referable to the organs of generation, by the facial expression alone. The description, however, is of importance to the beginner, because it indicates the value of detailed observation.

Since Duchenne has published his fine observations on the mechanism of facial expression, in his extensive and interesting work, a pathological facial expression is no longer an unsolved problem. In fact, such attempts have been made; for example, the excellent article of König on The Face in Tetanus. Since earliest times observations of characteristics have been at the disposal of the physician. Not to mention the facies Hippocratica of the ancients, every practising physician is familiar with the facies of a patient suffering from cancer of the stomach. The hysterical physiognomy is well known. The tuberculous expression (habitus) is recognised even by the laity, while a patient afflicted with a heart lesion is immediately spotted by the interne. Œdema of the eyelids may betray a Bright's disease, and this is but one example of many. Just as the insane asylum found its portrait-painter in Kaulbach, so might many diseases be recognised by a portrait of the patient.

However, no observations of this kind can be used as sole basis of diagnosis, but it may well furnish sufficient grounds for hazarding a tentative opinion. No sign is too unimportant, no symptom is to be underestimated, if the logical process employed by the diagnostician has as its foundation the discovery of symptoms of disease, and the observation of variations from the normal type. " Eum laudo," says Celsus, " qui quam plurimum percipit." As a matter of interest as well as of illustration, the following examples will show how a single glance at the face of the patient may prove of material aid to the clinician:

A patient complains of hoarseness; upon the forehead is an attached scar; his pupil is distorted. We at once think of syphilis, and the whole examination has a definite trend imparted to it.

Here is another case: A patient receives a Pott's fracture, and this is placed in a temporary splint. During the night our patient jumps out of bed, tears off the bandages, hops about on his broken leg, and it is only by good luck that the attendants arrive in time to force him back to bed without further accident. Had the evidences of alcoholism in the patient's face been noticed, delirium tremens would have been anticipated, and consequently a firmer bandage and more careful watching would have been ordered.

An old gentleman fractured his thigh while out walking, but not at the neck of the femur, as is usually the case with old people. The fracture was in the shaft. When v. Dumreicher was called in, he noticed a strabismus of minor degree. He immediately followed up this symptom, and on questioning found that the strabismus had existed fourteen days. He diagnosed: fracture due to carcinomatous degeneration of the bone, and also carcinoma at the base of the skull. Autopsy confirmed the diagnosis. The primary focus was a carcinoma of the parotid.

Naso-pharyngeal polypi (adenoids) are a disease of puberty, almost entirely confined to the male sex. Very frequently serious hemorrhages are caused by this condition. As the tumour blocks the naso-pharynx, the patient is forced to breathe with his mouth open. In many cases a glance leads one to suspect the true state of affairs, for the patient's physiognomy becomes peculiarly stupid and torpid. The anæmia, the patient's youth, and the nasal voice, are additional aids in diagnosis.

A patient presents himself with a swelling in the neck, which, at the first glance, proves to be a malignant tumour of the lymph glands. Where is the primary focus? The practised eye at once notices a scar on the lower lip. On inquiry it is found that this was the seat of an epithelioma removed by operation. The gland tumour is metastatic and due to the epithelioma.

An acquaintance of mine, about sixty years of age, came to see me. I noticed that his right eye protruded and was displaced toward the upper and outer side of the orbit. He told me that he was being treated by a specialist for a catarrh of the antrum. I at once suspected that the case was one of sarcoma of the superior maxilla, and examination confirmed this suspicion.

Careful inspection is of value not only when the patient first presents himself, but also after operation and in the course of surgical diseases. In describing

injuries to the skull it has already been emphasized that the sudden occurrence of strabismus or ptosis has an ominous significance.

A young and highly educated patient was suffering from anthrax of the nates. During the course of the disease shooting pains in the chest developed. The interne, who had charge of the case, diagnosed pleurisy. The complication might have been accidental, but on the following day symptoms of infiltration of the parenchyma of the lung developed—that is, pleuro-pneumonia. As no chill had occurred, the pneumonia might have been a septic one, but the general picture did not agree with this. On the following day jaundice was noticed. Small areas of consolidation appeared in widely separated spots. In spite of the absence of chills, the diagnosis of pyæmia was made, because of the signs in the lung and the jaundice. Autopsy showed that the lung was literally riddled with metastases.

A woman had a melanotic sarcoma of the trunk. During a morning visit I noticed drooping of one corner of the mouth. Facial paralysis. Evidently a melanotic deposit had formed upon the cortex. Other paralyses soon followed, then convulsions and death. The necroscopy showed tumours in the brain.

In a well-grown, buxom girl of twenty an inflammation of the left elbow-joint spontaneously developed. The posterior region of the joint grew doughy and spherically rounded. In spite of the blooming appearance of the girl, v. Dumreicher diagnosed tuberculosis of the joint, very early in the course of the disease, before any tumefaction had taken place. The girl was blond, had a transparent skin, and small opacities on both corneæ. As a child she had suffered from scrofulous ophthalmia. Later on the arm had to be amputated.

The number of examples which show how the observation of small details lead to the finding of new symptoms, and thus to the proper interpretation of the symptoms already observed, could be multiplied indefinitely. In each case we must notice the shape of the skull and face, the colour of the skin, the facial expression, the position of the eyes, the shape of the nose, the way the mouth is held, the speech, the action of the muscles, etc. The more we observe such facts the more we intensify and train our powers of observation. In this chapter we shall discuss only the diagnosis of diseases of the face. Diseases of the organs of mastication and of the mouth will be treated in the following chapter.

Let us begin with the disease of the face *par excellence*—Fothergill's neuralgia (Prosopalgia, or tic-douloureux).

The questions which the physician must answer, in order to make a complete diagnosis, are as follows: 1. Are we dealing with a true neuralgia of the trigeminus? 2. What is its distribution—i. e., what branches of the fifth are involved? 3. What must be regarded as the cause of the disease? As to the first point, the answer is not difficult; the second is harder to answer, and the third still more so.

Arguing from an anatomical standpoint alone, it will be evident that an inflammatory pain will be felt over the inflamed area. If a bone is affected, the pain is felt over the region of the inflamed bone. If a muscle is diseased, the pain is distributed along its course. If the connective tissue is inflamed, the pain will not correspond to the anatomical distribution of a bone, nerve, or muscle, but to the diffuse distribution of the connective-tissue planes. If a sensory nerve is affected the pain is felt along the course of all its branches. It is not limited by the anatomical boundaries of a bone or muscle, nor does it embrace the region of other nerves, as is the case in inflammations of the connective tissue. *Pain limited to the area of distribution of some definite nerve* is the first and most important symptom of neuralgia.

Clinical experience has furnished a second important distinction which aids in the diagnosis. It is generally known that inflammatory pain is lasting, and secondly that it is increased by pressure. In contradistinction to this, neuralgic pains are of a peculiar character. In the first place they are not lasting, but appear in *paroxysms*; in the second, only certain *points* are painful on pressure, not as in inflammation, where the whole inflamed area is sensitive.

And finally, clinical experience also furnishes a quantitative measure. In inflammatory processes the amount of pain has a fixed relation to the amount of pressure, the swelling, and other symptoms. In neuralgia the slightest disturbance, such as gentle pressure, opening the mouth, swallowing fluids, a loud word, can cause the most violent attacks. This may be followed at once by a return to the normal condition. There are cases in which pressure upon the nerves relieves the pain. We can therefore regard the following as characteristic of neuralgic pains:

Neuralgia is limited to the course of some nerve. It appears in paroxysms, and the pain is disproportionately severe. Neuralgic attacks, in addition, have an entirely characteristic train of symptoms seen in no other painful conditions. Among the symptoms which are seen in manifold combination may be mentioned hyperæsthesia of the skin in the affected region, anæsthesia in cases of longer standing, spasmodic contractures of isolated facial muscles, injection of the conjunctiva, increased lachrymation, and increased secretion of saliva and nasal mucus, etc.—a picture which is characteristic and not easily confused with any other disease.

If the pain is limited to the distribution of a single branch of the trigeminus, it is not difficult to decide on the branch affected.

On an anatomical basis the following varieties are differentiated:

1. *Supra-orbital neuralgia,* if the pain is distributed on the forehead, root of the nose, and the upper eyelid.
2. *Infra-orbital neuralgia,* with pain in the lower lid, alæ of the nose, cheek, and upper lip.
3. *Neuralgia of the superior dental,* with pain in the upper teeth.
4. *Neuralgia of the subcutaneous malæ,* with pain in the malar bone and region of the temples.
5. *Neuralgia of the inferior dental,* if the pain is felt in the lower teeth.
6. *Neuralgia of the lingual,* with pain in the tongue and mucous membrane of the mouth.
7. *Neuralgia of the auriculo-temporal,* with pain in the temples and the region of the ear.

These are the most frequent forms assumed by neuralgia limited to single branches. From an anatomical standpoint alone, it appears evident that a more extensive peripheral distribution of the pain corresponds to the involvement of more branches—i. e., a more central location of the disease.

Let us, for instance, suppose regions 2 and 3 simultaneously affected. The pain will then embrace the lower lid, cheek, side of the nose, and upper teeth. In this case we must suppose that not only the infra-orbital, but also superior dental is affected; or, in other words, the second branch, at a situation in which it has already given off its zygomatic twig, but not yet divided into its two terminal branches. If region 4 were also affected, the painful area would embrace the temple and malar bone as well, thus indicating that the subcutaneous malæ was also involved, or the trunk of the second branch before it gave off the zygomaticus; in other words, the whole second branch. Accordingly, with the help of anatomy, we can in each case map out the seat of the neuralgia by the peripheral distribution of the pain. On the face the three branches are indicated by the following three lines: A line drawn from the crown of the head along the anterior border of the parietal bone to the outer angle of the orbit, then continued to the inner canthus and to the tip of the nose, demarcates the region of the upper branch from that of the second. A curved line extending from the angle of the mouth to the outer canthus divides the distribution of the second branch from that of the third.

As a matter of fact, knowledge of the anatomy of the branching and peripheral distribution of the fifth nerve is indispensable if the seat of the neuralgia is to be determined. But this knowledge alone is insufficient, unless the fact that radiation to neighbouring branches may occur is also borne in mind during the examination. It must be determined whether radiation has occurred or not, and if it has, which branch is primarily diseased. This question may, as a rule, be determined by the following means:

1. The neuralgia begins in the nerve first affected; only later, in the course of the disease, does it reach the nerves affected by irradiation. 2. During *each* attack the primary nerve is painful; the pain often fails to appear in the contiguous nerves. 3. Each neuralgic at-

tack begins in the primary nerve; only at the height of the paroxysm is pain felt in adjacent nerves, and it disappears from these before the end of the attack is reached. 4. The irradiated pain is less intense.

Valleix's puncta dolorosa—the painful spot at the point of exit of the nerve-trunk—is of such doubtful value in diagnosis, and is still so differently interpreted by various authors, that we will not enter into its discussion.

If we have been able to determine whether irradiation takes place or not, a new question arises: Is the neuralgia peripheral or central? Unfortunately, our knowledge is not as yet sufficient to answer this question. If the neuralgia is limited to a single branch, and is due to some peripheral cause (trauma, sudden cooling of the skin), it is fair to assume that the neuralgia is peripheral. If, in addition to the neuralgia, other symptoms of brain trouble present themselves, the opposite conclusion is more reasonable.

To affirm more than this would oblige us to strain the truth, and even this assumption, if we remember the insufficient data, may not be justified.

In answer to the third question—namely, the cause of the neuralgia—only a few suggestions can here be given. Neuralgias of intermittent type—especially supra-orbital—are often due to malarial infection. Here quinine gives brilliant results. As a rule, it is necessary *ex juvantibus* to come back to the etiology. Sufficient cause for neuralgia exists in a carious tooth, a foreign body embedded in a bone, a scar, a tumour, or an inflammation of the bone through which a nerve passes. If such local causes are lacking, a trauma previously sustained, some very sudden change of temperature, mercurial poisoning, etc., may be the causal factor. Finally, hysteria, anæmia, some distant disturbance (liver trouble, any abdominal disease), must not be forgotten, and, in some instances, the effect of treatment may serve to clear up the etiology.

CHAPTER VI

SURGICAL DISEASES OF THE ORBIT, NOSE, AND FRONTAL SINUS

In this day of specialists but few diseases of the orbit reach the hands of the general surgeon. In consequence this discussion will be quite fragmentary.

Orbital *Blood Effusions* are of common occurrence. Formerly it was believed that subconjunctival effusions, appearing several hours after an injury, invariably signified a fracture of the roof of the orbit. More careful investigation has, however, shown that such ecchymoses may arise from rupture of the orbital vessels. Their source may be found within the skull, the blood reaching the orbit either through the optic foramen or through the sphenoidal fissure. It follows that exophthalmos or lateral displacements of the bulb subsequent to an injury, and combined with subconjunctival ecchymoses, can not, without further corroboration, be regarded as a proof of fracture of the orbital roof. But in fracture of the base this assumption is, as a rule, justified, for the cause of the effusion usually is due to a direct extension of the fissure at the base into the roof of the orbit.

If subcutaneous emphysema accompanies these symptoms, and the existing exophthalmos is increased by sneezing, it may be positively stated that the inner or lower wall of the orbit has been completely broken through, and communicates directly with the nasal cavities. Effusions into the lid are due to rupture of the vessels situated anteriorly to the tarso-orbital fascia, for this structure opposes the advance of blood effused behind it.

Deep penetrating wounds, if infected; strong caustics, especially in solution, which reach the peribulbar cellular tissue through a tear, often cause rapid sloughing of the orbital cellular tissue, the acute *Orbital Abscess.* Such an abscess may also develop in the course of erysipelas, or may be due to pyæmic metastases.

The gross characteristic symptoms of this affection are swelling of the conjunctiva (chemosis), œdema and reddening of the lids, exophthalmos, the unilateral distribution of the swelling, and pain induced by pressure upon the bulb.

In this process the orbital cellular tissue is the seat of the inflammation. It is therefore easily understood that the bulb is crowded directly forward. Numerous deep incisions, extending through the torso-orbital fascia, must be made early in order to afford free drainage.

There are cases in which the symptoms of acute orbital abscess are present, but in which the displacement of the bulb is not directly forward, but toward one side. If the process is of spontaneous origin in an individual still in the period of growth, we must think of an idiopathic diffuse osteomyelitis. It is true that this disease is very rare. I have seen only three cases. The diagnosis was easily made. The disease began with high fever (chills); appearance of the lids as seen in gonorrhœal ophthalmia (unilateral); widespread œdema over the frontal bone; early fluctuation at two separate points without any intercommunication of the fluctuation; and bare bone upon making the incision. In the one case I was able to diagnose an abscess of the frontal bone itself. On trephining, the pus flowed out with a pulsating movement. Evidently the internal periosteum (dura) was dissected away from the bone, and the abscess, which pointed toward the skull, communicated by an opening with the one situated between the diploe. In one of the three cases osteomyelitic foci appeared at other sites (in the legs).

Chronic abscesses, which occur in the orbit in connection with bone tuberculosis, naturally do not displace the bulb directly forward because they are inclosed by a pyogenic membrane, and do not break into the diffuse cellular tissue of the orbit. They may point spontaneously into the conjunctival sac or may open externally. At times the bone abscess is situated at the margin of the orbit in such a way that the pus can point only anteriorly to the tarso-orbital fascia—that is, through the lid itself.

The situation of an ORBITAL TUMOUR may be determined by the same method of deduction used in connection with orbital abscesses—the displacement of the bulb. Tumours situated behind the bulb crowd it directly forward. Tumours situated to one side crowd it to the opposite side. If the bulb is displaced forward and to one side, the tumour is situated partly to the side and partly behind the bulb. The tumour may be free in the orbital cavity and movable (myxoma, cylindroma, angeioma), or arise from the bone (sarcoma or osteoma of the frontal, sarcoma of the superior maxilla), or originate from the neighbouring pneumatic spaces (tumours of the antrum of Highmore or of the frontal sinus).

It is important to judge the position of the bulb correctly when dealing with tumours of this region. If, for instance, the case is one of sarcoma of the superior maxilla, and exophthalmos is present, this condition informs us that part of the tumour must have spread behind the bulbus oculi. We are thus enabled to form some conception of its growth among the deeper tissues, which are beyond the reach of direct investigation.

Another symptom requires mention in the case of malignant tumours. If a tumour be benign, the movements of the bulb may be interfered with, but only in such wise and degree as is accounted for by purely mechanical causes, which are readily deduced from the size and position of the tumour. Malignant tumours, however, involve all tissues, so that very early the action of at least one muscle is completely inhibited. In consequence all movements of the bulb toward this side are entirely destroyed. If the tumour involves the bulb itself, the eyeball becomes completely fixed at an early stage.

The organ of sight requires careful observation on the part of the surgeon, even in distantly situated conditions. Ptosis and strabismus, occurring in meningitis, have already been mentioned. Lagophthalmos occurs in facial paralysis. In injuries of the cord and sympathetic, malignant tumours of the parotid gland, affecting the sympathetic, etc., the behaviour of the pupil and the position of the bulb, require careful consideration.

Various forms of ulcers appearing on the *nose* offer difficulty in their diagnosis. Cancerous, lupous, and syphilitic nodules, and the destruction of the nose consequent to them, are, as a rule, readily distinguished one from another. Of the *Epitheliomata*, those met with on the nose are usually of less than ordinary malignancy, and clinicians have chosen a special name for them, which is still partly in use—ulcus rodens, Schuh's flat carcinoma, epithelioma. Epithelioma, if well advanced, is recognised, as a rule, as a superficial ulcer, consisting of an irregular, flesh-red, shiny, ulcerating surface, which is studded with small nodules. If the growth has not been smeared with irritating ointments it is covered by scanty thin secretion, and the floor of the ulcer is quite as hard in consistence as the narrow raised margin, which is composed chiefly of small, isolated nodules. In addition to the fact that this growth commonly occurs only in people advanced in years, we must further notice that neither lupus nor syphilis have so slow a course. In these slowly progressing epitheliomata one peculiarity, which is extremely misleading to the inexperienced, must be especially mentioned. This is the occurrence of a pellicle of skin in isolated spots over the ulcerating surface. This deceptive symptom must not be allowed to interfere with early operative treatment.

The spots of epidermis are really nothing but hardening of the epithelial cells of the cancer, a sort of pathological distortion of the normal formation of epidermis. The older physicians erroneously regarded it as a healing process, and used a variety of remedies in the vain endeavour to help Nature in her conservative efforts. Quite wrong! In fact, this statement applies as well to epitheliomata in other regions. If the ulcer runs a more malignant course from the very start, or gains greater malignancy after it has existed for some years, it does not differ clinically from epithelioma elsewhere. Epitheliomata occurring inside the nose are more malignant than the rodent ulcer. We must not forget that villous cancer, which originates in the antrum and has escaped observation during its development, may first attract attention when it appears in the nasal cavity.

Lupus of the nose should be readily recognised. There are small nodules, the size of a pin-head, which are seen through the thinned and shining skin, and have a bluish-red colour. They are isolated or strung along curved lines. The nodules may be larger, covered with scales, and in this situation usually lead to ulceration (lupus exulcerans). Though the process often causes a rapid and excessive formation of small nodes (lupus hypertrophicus), it finally ends by marked destruction of the tissues. The process spreads to the mucous membrane of the nose—not infrequently perforating the cartilaginous septum—and exceptionally involves the mouth and gums as well. In addition to these changes, flat or irregular scars are commonly found at other spots; these can in their turn become the seat of new lupous eruptions. If we keep this picture in mind, and further remember that, as a rule, youthful individuals are attacked by the disease, a mistake is not readily made.

Syphilitic Gummata of the nose and the ulcers which develop from them may, in some instances, offer difficulty in their diagnosis. If they occur in youthful subjects, afflicted with hereditary syphilis, they may be mistaken for lupus; in older individuals they may be confounded with epithelioma. If signs of concomitant or previous syphilis appear elsewhere on the body, all difficulty is removed; if, however, such signs are wanting, as in hereditary syphilis, the nature of the nodules or, even more, the ulcers springing from them, are sufficiently characteristic to permit of diagnosis. The gummata are larger, separately placed, and soft (elastic). The epitheliomatous nodules are small, firm, and aggregated so as to form a flat, sharply circumscribed surface surrounded by a ridge. The lupoid nodules may be large and soft, or small and joined closely to one another; but, in either case, we find the characteristic isolated and diffusely scattered nodules, which have a bluish-red colour and shine through the thinned-out epidermis. Syphilitic ulcerations of the nose are frequently followed by destructive processes in the nasal cavity—unlike lupus, destruction of the bony septum is commoner—or in the other neighbouring cavities. Syphilitic ulcers at times heal up on one side, while the opposite edge shows a new infiltration, which is constantly breaking down. This form of partial healing causes an ulceration known as *serpiginous*.

The rare disease *Rhinoscleroma* appears in a platelike infiltration of the septum and ala of the nose, and of the upper lip, which has a characteristic, almost cartilaginous, consistence. It does not ulcerate, even after a long period, and progresses slowly in spite of all treatment.

The most frequent tumours occurring in the nasal cavity are MUCOUS POLYPI. Strange to say, they are frequently mistaken for other growths. For instance, small, round, translucent, grayish abscesses appear on the septum as the result of perichondritis. These may, upon superficial examination—and this can not be denied—appear like the rounded end of a polyp. But the diagnosis should be made by means of the broad base attached to the *septum*, their rapid growth, and especially, if it exist, their symmetrical occurrenc on both sides of the septum. If opened, they discharg pus. In children a smooth, round, foreign body, if hig up, may deceive us. To guard against this error em ploy the sound. Beginners frequently mistake for pol pi those thickenings which appear on the lower edge the greater turbinated bone. These swellings of th mucous membrane covering the turbinated bone ar red—not gray or grayish-yellow like polypi—or dar red if of venous origin. They are situated laterall are sessile, not rounded in shape, and of much harde consistence.

The number of nasal polyps which may be present i unlimited, but, however numerous, the nose itself i never distended by them, and the nasal bones are neve pushed forward. Formerly I thought this rule withou exception, but after hundreds and hundreds of observ tions I have seen such exceptions. *Exceptio firmat reg lam!* If one side of the nose is found larger than th other, the one nasal bone pushed up, the nostril of thi side markedly enlarged and more distorted than th other, we must be prepared for something other than nasal polyp. As a rule it is a sarcoma, usually a ver soft sarcoma growing down from the base of the skull These commonly appear in individuals of riper years, bu I have seen them in children. The malignant nature o the doubtful growth is further confirmed if some spo of the skin of the nose shows an area covered by a fin star and netlike distribution of blood-vessels, which oc cur also over malignant neoplasms of the skin in othe regions. In these cases one or more lymphatic gland of the submaxillary region are usually enlarged.

If the just mentioned indications of a malignant neo plasm of the nasal cavity are present, we must never fail to examine the nasopharynx, to carefully test the position of the bulb, and to make sure that no symptoms which would indicate that the growth originated on the basis cranii escape our notice. These symptoms will be referred to later.

In passing it may be mentioned that herniæ cerebri have prolapsed into the nasal cavity, and that their smooth, rounded surface caused them to be mistaken for polypi.

The *Frontal sinus*, the continuation of the nasal cavity, has, especially in recent days, received much attention from the surgeon. Its diseases become manifest only when distention of the cavity—that is, a swelling—appears. This distention of the frontal sinus causes not only an increased bulging of the brow, but also a swelling in the orbit. In children this swelling appears along the nasal side of the orbit, and at times, as the condition increases, results in an outward displacement of the bulb. In adults the roof of the orbit may bulge, and in some instances the bulb may be crowded downward. The causes of this displacement are blood effusions (very rare), empyema, hydrops, and neoplasms. The diagnosis can be made only when we are dealing with an acute inflammatory process. An empyema is ushered in by severe symptoms—chills, delirium, strabismus. Sometimes the pus points into the interior of the skull and causes a fatal outcome. Diagnosis from a neoplasm can not be difficult, but from inflammatory conditions in neighbouring cavities, especially if the course is less acute, the differentiation may be hard to make. For instance, when the process breaks into the orbit, it may produce the picture of a periostitis of the orbit. Of more use than further hints is the following case, in which Strohmeyer made the diagnosis through logical deduction. The case was doubtful. Strohmeyer diagnosed empyema of the frontal sinus by the severity of the onset, the violent pain on one side of the head, and the dry condition of the same side of the nose. The diagnosis was confirmed when blood and pus spontaneously discharged from the other nostril, for rupture had taken place into the sinus of the healthy side.

CHAPTER VII

DISEASES OF THE MAXILLÆ AND OF THE TEMPORO-MAXILLARY ARTICULATION

TRAUMATA of the inferior maxillæ are so readily open to inspection that it is rare for any difficulty in diagnosis to arise. Inspection alone is sufficient, and even the laity, as a rule, know whether the bone is fractured or not.

In dealing with a *fracture*, the steplike interruption in the continuity of the teeth, the abnormal mobility, the tear in the gum and mucous membrane, and the inability to bite, are so evident that any problem of differential diagnosis need not be considered.

In reference to *dislocations* of the temporo-maxillary joint few points require discussion. This injury, which is caused by opening the mouth too wide, is the only traumatic dislocation which occurs without rupture of the capsule. The condyle, still within the capsule, is displaced in front of the eminentia articularis, and is unable to return. A symptom, which is seen only in this injury, at once results, the patient is unable to shut his mouth. This is pathognomonic.

The sudden inability to bring the jaws together renders the recognition of this dislocation so easy that a Bohemian surgeon was wont to jump up and rush with threatening fist toward any patient entering his office with his mouth characteristically open. The sudden fright caused the patient's dislocation to reduce spontaneously.

The dislocation may be unilateral or bilateral. In either case it is readily recognised. If the dislocation is bilateral, a vertical line dropped between the first incisors of the lower jaw will fall in the median line of the body. If it is unilateral, the upper end of the line will incline toward the affected side. If the patient has lost his incisors, or has no teeth, we notice that the affected side of the lower jaw is farther removed from the upper jaw than the healthy side; or, in other words, that a plane drawn through the edge of the maxilla is not horizontal, but inclined downward toward the diseased side. Examining the neighbourhood of the joint, we find a hollow at the site of the joint, and the condyle is found located anteriorly. At the same time the dislocated jaw is protruded, the chin appears longer than normal, the patient salivates, and speech is interfered with.

Dislocation of the jaw is characterized by inability to close the mouth, and this, as already stated, is pathognomonic of this condition. The opposite condition, inability to open the mouth, *Lockjaw* (ankylostoma), is a symptom common to several diseases. An uncommonly rare condition, as yet not verified by autopsy, but well authenticated on the living, is a backward dislocation of the condyle, which presupposes certain structural abnormalities in the articulation. It occurs in female subjects, and manifests itself in a sudden and unexpected inability to open the jaws. The patient makes various, often forcible, efforts to open her mouth. Some sudden jar occurs, and the jaw is again movable. Most commonly lockjaw appears as a symptom in periostitis of the lower jaw resulting from caries of the last molars. Violent toothache, swelling of the cheek, and then lockjaw—this is the typical, recurring picture seen in thousands of cases. Observation shows that the pain ceases when the swelling appears. The same picture may be due to the eruption of a wisdom tooth. Consequently, in individuals of the proper age, this process must be kept in mind. Phlegmonous inflammations of different kinds occurring in the neighbourhood of the articulation, or even in the region of the ramus, can cause lockjaw; for instance, a phlegmonous angina. In all these cases we deal with an acute process which can be deduced by its other symptoms. The cause of the trouble is readily recognised if due to cicatrices, inside the mouth, resulting from a former stomatitis. Quite as simple are the cases in which cancer, extending from the upper to the lower jaw, or vice versa, acts as an inelastic band and impairs movement.

It is evident that lockjaw can result from inflammations of the masseter. I saw such a case due to a stone striking the cheek. The muscle is not only tense and hard as a board, but also painful, even in the spots not covered by the outer contusion, especially to palpation from within the mouth.

If no extra-articular causes are present to account for the lockjaw, an inflammation of the joint itself should be looked for. Sometimes, after scarlatina, inflammation of the articulation takes place. Occasionally caries of the petrous bone advances close to the joint. In such cases sensitiveness in the neighbourhood of the joint can be demonstrated. This, combined with the contracture of the muscle, and the absence of other causes, is sufficient to assure a diagnosis.

The jaws are greatly exposed to *acute inflammations*, because they form the bed of the teeth, which are so often carious. But among operatives engaged in the manufacture of phosphorus matches the jaws are even more exposed to insults. The direct influence of the phosphorus fumes upon the jawbones is marked by a peculiar periostitis. It is characterized by the growth of a thick callus (due to rapid proliferation of the connective tissue) on the outer surface of the periosteum; on the inner surface new bone is produced in scattered spots of considerable extent. Pyogenic cocci make their entrance at the same time, so that an ossifying and suppurative periostitis go hand in hand, but at different spots. The lower jaw soon necroses. It then appears embedded in a capsule which is roughened externally and bony in spots internally, yet separated from this capsule by surrounding masses of pus. The teeth drop out at the beginning of the process, the gums then retract, so that the alveoli are laid bare, and appear like cavities filled with pus. The thick callus is perforated here and there by fistulæ which allow the pus to escape. Naturally these changes take place only slowly, and accompanied by great pain. Sinuses at the lower margin of the jaw result, discharge pus freely, and lead directly to bare, necrotic bone. In the upper jaw no discharge toward the exterior occurs, as far as I know; nor is the swelling as extensive as in the lower jaw. At the boundary of the visible necrosis the teeth are loose, and this symptom shows how the outer swelling and the necrosis advance hand in hand.

Diagnostically, PHOSPHORUS NECROSIS is distinguished from phlegmonous periostitis of the jaw, following caries of the teeth, by the whole symptom-complex.

I consider it of importance that in phlegmonous periostitis, which often follows the extraction of a tooth, the necrotic bone is never visible to such an extent, nor the gums so retracted, as in the variety due to phosphorus.

Beginners might be embarrassed by a large ulcer, which primarily involves the cheek, spreads to the floor of the mouth, and then attacks the lower jaw. In such cases, a necrotic part of the lower jaw may be visible at the centre of the ulcer; for instances are on record in which an epithelioma takes its origin from the cheek or floor of the mouth, and then, advancing to the jaw, involves the bone, producing a sequestrum. I have seen several such cases. Beginners are, as a rule, hasty in advancing a judgment and diagnose necrosis. But in necrosis much pus is produced; there is increased periosteal bone production, which forms an involucrum about the sequestrum. The break in the continuity of the tissues is not large, and the granulations are soft. In epithelioma the ulcerating surface is extensive and has hard edges and an indurated base; production of pus is lacking, and no lockjaw occurs. Necrosis is, of course, also present, but epithelioma is the primary disease.

Another tráde disease was first noticed and described in Vienna. This is the so-called mother-of-pearl-workers' ostitis, or CONCHIOLIN-OSTITIS, which spares the upper but not infrequently attacks the lower jaw, and many other parts of the skeleton. It is characterized by an enormous thickening of the bone, which gradually disappears in the course of a few weeks without leaving any trace of its presence. Suppuration very rarely occurs. As a rule, the thickening is unaccompanied by any change in the soft parts, the skin is not reddened, and the adjacent soft parts look normal. On the lower jaw the process travels downward from the neighbourhood of the articulation, but spares the joint and the coronoid process. Lockjaw (ankylostoma) is absent. From the above it follows that the diagnosis can lie only between some central bone tumour, which enlarges the bone, and the above disease.

But conchiolin-ostitis is accompanied by severe pain, is more rapid in its course than a benign neoplasm, and extends downward along the bone.

Another peculiar disease also has its favourite site in the region of the jaw, especially of the lower one. This disease is ACTINOMYCOSIS. Undoubtedly the ray fungus gains entrance next to the teeth, or through an empty tooth-socket, and from there works into the soft parts. Once established, it spreads and causes a labyrinth of sinuses in the soft parts. It also attacks the bone, producing fistulæ and necrosis. Externally, while the process is developing, we see abscesses, ulcers, and sinus formation. Its spontaneous appearance, absence of all pain, and frequently the violet hue of the overlying parts, remind us of tuberculosis. Actinomycosis was, as a rule, mistaken for tuberculosis (scrofula) as long as the disease was not understood. The trouble is diagnosed by means of the following symptoms: The foci are very numerous, but appear only on the cheek and one side of the neck, although no tuberculous processes can be found in the rest of the body. Swelling of the lymph glands is prominent by its absence. The healthy appearance of the patient, absence of hereditary taint, and the peculiar yellow granules found in the pus speak against tuberculosis. Finally, the microscope confirms the diagnosis by demonstrating the fungus (Fig. 3).

At times an acute form of actinomycosis is found, and presents the picture of a phlegmonous inflammation. In such cases the diagnosis can be made only by examination of the pus.

In dealing with *Neoplasms* of the jaws, the upper and lower maxillæ require separate discussion.

In the lower jaw the different course of periosteal and myelogenic (central) tumours is often striking.

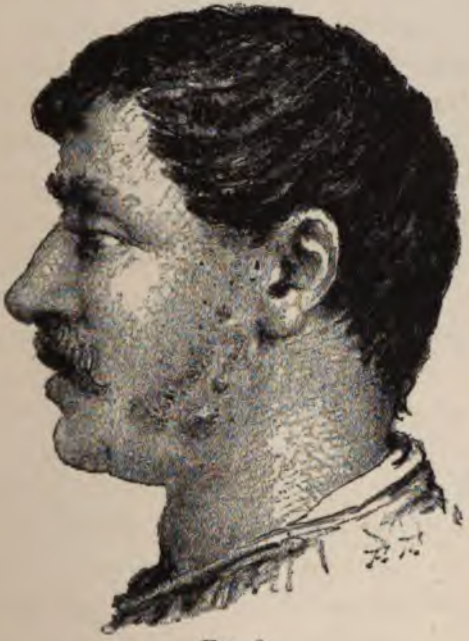

FIG. 3.

The central tumours expand the bone, the surfaces of the maxillæ become convex and the edges rounded off. As the expansion continues, the tumour not only grows prominent externally, but also in the direction of the oral cavity. The bone, which has attenuated and softened, becomes a mere shell, which produces a parchment crackling on palpation. Even this shell can disappear in spots, so that gaps are to be found. The tumours which give rise to these changes, as a rule, are soft sarcomata. They develop rapidly in the course of a few months in young adults and children. The lymph glands are involved comparatively late. The presence of a perforated or partially destroyed bony shell is conclusive. *Periosteal tumours* rest upon the bone. They are consequently prominent only on the outer surface, while that part of the surface of the maxilla which faces the oral cavity shows no bulging. The lower margin of the jaw may retain its sharp edge until the tumour advances to it or begins to extend over it. The case is less clear if the edge of the bone is involved by the growth, and consequently appears rounded. Here the absence of swelling on the inner surface is a strong, or rather a decisive, argument in favour of a periosteal tumour. But a periosteal growth may involve both the outer and inner surface, and the edge of the bone, so that the bone is all but surrounded by the growth, yet remains intact in its centre. The lack of the egg-shell crackling on the surface of the tumour and the absence of any trace of a bony shell are conclusive.

In the upper jaw we must distinguish between *tumours of the antrum* and of those of the *maxilla*. A tumour of the antrum grows in the direction of least resistance. It consequently penetrates into the nasal fossa, presses the thin wall of the canine fossa forward, raises the floor of the orbit upward, and thus crowds the bulb upward and outward. After occupying the nasal fossa, the growth may proliferate and appear through the choanæ, though the thick lower wall of the antrum still remains intact.

In distinction to antral tumours, bone growths involve the alveolar process, destroy the teeth, and form an ulcerating mass of considerable size upon the surface of the maxilla. In addition, they attack the hard palate, causing it to arch forward, while the bulb retains its normal position and the canine fossa is not obliterated.

Cases in which all the signs and symptoms of an antral tumour were present, but in which sections of the extirpated maxilla showed that the tumour originated from the bone, do occur. Here the growth started in the thin diploic layer found between the two bony lamellæ of the anterior wall of the antrum. During its growth the tumour penetrated the antrum, and then continued to grow unhindered, with all the symptoms of a true antral tumour.

We frequently find the canine fossa pushed outward by a rounded swelling, which is sharply circumscribed and painless. It enlarges but slowly, and fluctuates. Formerly, physicians were satisfied to diagnose *hydrops antri Highmori*. To-day, we know that we have to deal with one of three conditions: with a dentigerous cyst, chronic periosteal abscess, or cystic polypus of the antrum. A hydrops of the antrum, in the strict sense of the word, does not occur. Only a muco-purulent or purulent collection of fluid (empyema) can accumulate. If the above-mentioned symptoms occur a different condition exists.

In all of these conditions the bony wall, which bounds the swelling anteriorly, may grow so thin that it can be pressed inward like parchment.

Dentigerous cysts develop toward the upper jaw by an abnormal growth of the permanent teeth from the enamel sac, which then often incloses the unerupted tooth. There are also dental cysts which arise from the root of the tooth without any abnormality of development.

Chronic subperiosteal abscesses are still rarer. After they exist for a long period (I saw one case of thirteen years' duration) they assume an appearance which in no way resembles an ordinary abscess, for the periosteum, which covers the abscess, is stimulated to bone formation, and the newly formed lamellæ are then thinned out. The parchment crackling is wrongly ascribed to the anterior wall of the maxilla. In these abscesses the contents usually is serous, at times mucous. The swelling is not painful.

The diagnosis of dentigerous cyst is justified if one or more of the permanent teeth have failed to develop and a slowly growing cyst occupies the resulting gap.

If these signs are wanting, the diagnosis can be made only at the time of operation, for after the swelling has been split open, in the case of a mucous polyp, polypoid masses are encountered; in case of a dentigerous cyst, the unerupted tooth; in the subperiosteal abscess, pus. In none of the conditions does fluid injected into the cavity of the cyst flow out of the nostrils. In former days such findings would have led to the diagnosis of hydrops of the antrum. If fluctuation and parchment crackling were present the diagnosis was supposed to be assured. Their conception of the condition was, that in hydrops the outlet of the antrum was blocked, and that the secretions therefore accumulated. Such a condition has never been verified by unmistakable anatomical findings.

Professor Zuckerkandl's exhaustive researches demonstrate that cysts of the maxilla show great diversity in their behaviour. In general we distinguish between external and internal. The external cysts crowd out the anterior, at times the posterior, or even lower wall of the bone. They project into the vestibule or into the oral cavity. If the cyst grows as far as the floor of the antrum, it raises this upward, and thus encroaches upon its cavity. In a similar manner the floor of the nasal fossa can be raised up and thinned out. Such cysts have been known to burst. Cysts of the intermaxillary bone show an especial tendency to grow toward the nasal fossa or toward the gums. Empyema is due either to affections of the teeth and periosteum or to a purulent rhinitis. Consequently the anamnesis varies greatly. In regard to cysts, Zuckerkandl emphasizes the following: Arching forward of the anterior portion of the alveolar process or of the outer wall of the alveolus, together with parchment crackling, speak for cyst. If the internal wall of the antrum bulges in the middle meatus of the nose without distortion of other parts, the symptom indicates empyema. If the *inferior* and the *middle* meatus of the nasal fossa are encroached upon, the diagnosis of empyema still holds good.

Analogous conditions likewise arise on the lower jaw, except that mucous polyps are naturally excluded. The case may either be dentigerous cyst or chronic subperiosteal abscess. With these two alternatives kept in view, the reasoning is similar to that employed in like conditions of the upper jaw.

The direction in which *retro-maxillary tumours* enlarge is peculiar. They advance around the outer surface of the upper jaw, make their appearance below the masseter, below the zygoma, in the temporal region, even in the mouth, and in the orbit, where they may crowd the bulb out of place. But the walls of the antrum do not bulge, the alveolar process is not thickened, and the gum not crowded downward. On examining the swelling we are forced to the conclusion that it is soldered upon the jawbone, if this expression is permitted. It may be, and has been, mistaken for a tumour of the parotid.

CHAPTER VIII

DISEASES OF THE MOUTH AND PHARYNX

EXAMINATION of the condition of the mouth is very frequently made, and often proves of great value. The internist sees in a herpes labialis a confirmation of his diagnosis of typhus; the surgeon regards an edematous upper lip seriously when called on to decide whether or not a child has a scrofulous diathesis. The gums are to be examined in all cases of suspected stomatitis, scurvy, and anæmia. Such an examination once saved me from a serious mistake. A child with hare-lip was to be operated upon. I examined the gums and saw by their condition that two teeth were about to appear; I therefore advised the parents to bring the child at some later date. During the period of teething the child was seized with convulsions. When I was informed of this, I considered it advisable to wait until the child was still older. The parents, however, went to another surgeon, but concealed the condition. The surgeon operated on the hare-lip; during the following night convulsions occurred, and by the next day the child was a corpse. The whole mucous membrane of mouth and pharynx must be carefully examined if syphilis is suspected, for the epithelium of the tongue often betrays the disease in spite of the obstinate denials of the patient. "*Labia custodiunt scientiam lingua loquitur* *judicium.*" In practice the physician must act with circumspection if he finds the signs of syphilis. If they are noticed, it is unnecessary to tell the patient, for a false imputation may convert a hot-headed patient into a harmful enemy.

Hardness of hearing can frequently be cured by tonsillotomy; therefore, in this condition one must never fail to examine the mouth. In swelling of the cervical lymph glands, as we will see later, careful examination of the mouth will often demonstrate the local point of infection. Such examples are very numerous. The coated surface of the tongue requires at least cursory mention. We must agree with Strohmeyer, that the world-wide custom of inspecting the tongue at each visit should not be abandoned; for, in one case in a hundred, or oftener, it leads to the discovery of some disturbance. For a surgeon, it is true, it is more important to examine and to be able to judge the condition of the wound. Yet it is a satisfaction to see the abnormal conditions of the tongue, such as dryness or coating, disappear in conjunction with the improved condition of the wound, of the findings shown by the thermometer, and the composition of the urine. In those cases of erysipelas which are ushered in by so-called gastric disturbances, by nausea, and heavy brown coating of the tongue, v. Dumreicher prescribed an emetic as a matter of routine. I can give the assurance that in all such cases the patient was greatly relieved. In these days of laparotomy we always examine the tongue after operation; it tells us a great deal.

When we turn to the diagnosis of local conditions, we first of all think of the common *ulcerations* of the mouth.

A practical classification of ulcers of the mouth consists in dividing them into fetid and non-fetid cases; for fœtor ex ore is so noticeable a symptom that it at once turns the thoughts of the physician into certain channels.

An equally practical classification is regional. Secondary syphilitic ulcerations appear on the palate, likewise diphtheritic gangrene; scorbutic ulcerations and cancrum oris on the gums; noma and malignant pustule on the cheek; carbuncle only on the lip; aphthæ scattered upon the mucous membrane of the cheek, floor of the mouth, and gums.

But it is still more practical to divide the ulcers, according to their chief symptoms, into certain groups, because these symptoms at once exclude ulcerations belonging to other varieties.

If, in the course of a scarlatina, a white membrane appears on the tonsils, palate, fauces, and uvula, and on the third or fourth day becomes gangrenous, accompanied by an extremely fetid odour, marked swelling of the cervical glands, and severe constitutional symptoms, the condition must be DIPHTHERIA. The same disease may be primary, without scarlet fever.

The milder form of the disease, *diphtheritic tonsilitis*, which produces milder general symptoms, is characterized by small white patches on the tonsils. These two conditions must not be confounded with that form of angina known as FOLLICULAR ANGINA, or herpes of the tonsils. The constitutional symptoms may correspond and the swelling of the glands also, but the appearance of the tonsils can be similar only at the onset. In follicular angina the tonsils are swollen, reddened, and studded with numerous yellowish dots. Strohmeyer says: "The tonsils look like the star-studded heaven." These yellow plugs consist of pus, mucus, and bacteria which block the lacunæ of the tonsils. They correspond in size, number, and regularity of distribution to the situation of the lacunæ, and therefore appear only on the tonsils, and are strictly limited to the lacunar openings. Croupous membranes, however, have no definite distribution or size. It is decisive if they occur on the fauces, soft palate, and uvula.

Another group, which can not be brought into any relation with the other ulcerative processes, is characterized by a large, very tense infiltration of the surrounding parts, and usually appears as a gangrenous condition of the cheek in the neighbourhood of the corner of the mouth. The widely distributed, boardlike infiltration is apt to call up the picture of anthrax. But this disease need not be discussed here, for it can never be properly counted among the ulcerations of the oral cavity. It arises in the skin, and then spreads to the subcutaneous tissues, but as it never perforates, it causes no ulcer inside the mouth. Malignant pustule, likewise, which appears on the exposed portions of the skin, can not be discussed here, although, in passing, we may state that the disease begins with rapid pustulation and formation of scabs, which are depressed. The area is odourless and surrounded by a circle of vesicles. The condition is followed by swelling, which, however, does not lead to perforation.

The diseases referred to are noma and advanced cases of mercurial stomatitis. These begin in the mouth, and may lead to great destruction of the soft parts of the cheek, of the gums, and bones. They are distinguished from one another in the following manner:

Noma appears only in children, and then only after severe, debilitating diseases—following small-pox, scarlatina, typhus, cholera, etc. The word *after* is to be emphasized, for, in other words, noma appears only during convalescence, and then suddenly, extending below to the upper or even lower lip, and reaching above to the lower eyelid. The cheek is swollen, waxy, pale in colour, and shining like a fatty surface. On the inner side of the cheek is a small gangrenous ulcer, corresponding to which there is a hard spot of infiltration in the outer swelling. A rapid gangrenous destruction of the whole thickness of the cheek starts from the ulcer and extends to the gums, and even to the alveolar processes, so that a large hole, which communicates with the mouth itself, is formed. *Mercurial Stomatitis* is recognised by the absence of an antecedent severe disease, by the positive knowledge that mercury has been taken in large quantities, and especially by the fact that the skin remains intact, while it may be destroyed in a short time, even in two or three days, in noma. Noma in most cases is a fatal disease. The patient's strength fails; delirium, profuse diarrhœa, and œdema of the feet precede death. Mercurial stomatitis, on the other hand, is a very slowly progressing affection, and only exceptionally does it prove fatal.

Two other conditions have common characteristics and a superficial resemblance. These are scorbutic affections of the gums and aphthous stomatitis. Both processes run an afebrile course, begin in the gums, cause a foul odour, swelling, softening and pain of the gums, and loosening of the teeth. But they are readily differentiated. In SCURVY the gums are dark, bluish, and greatly swollen. They rise above the crowns of the teeth and bleed spontaneously or on the slightest pressure. The rest of the body shows signs of scurvy. In *stomatitis* the gums are reddened, and bordered by a markedly yellowish edge, consisting of a softened pulp, which ulcerates and exposes the roots of the teeth. An exact impression of this yellow edge is found on the mucous membrane of the cheek corresponding to both the upper and the lower jaw. The mucous membrane of the cheeks and the lips are swollen, the tongue is coated. The affection rapidly disappears on using potassium chlorate, sometimes within two to three days.

Very small ulcers, with sharply demarcated edges and yellowish base, are seen, especially in the mouths of women. They often recur regularly at the menstrual period, and may lead to the suspicion of venereal disease. This suspicion is all the more readily accepted by the inexperienced if the same condition is found on the genitals, as occasionally may happen. Under these circumstances we must be politic and circumspect. The small ulcers may well be innocent *Aphthous* spots. They are recognised by their extreme sensitiveness, which annoys the patient in speaking, chewing, and laughing. They are further characterized by their bright-red border, and especially by the fact that they disappear in the course of a few days, without treatment.

Children also suffer from infected ulcers under and at the tip of the tongue. They may appear suspicious, for, in addition to their lardaceous base, the edges may be infiltrated, and healing proceed very slowly. In spite of this, they are innocent, and related to the process of dentition.

On the tongue two pathological conditions are of interest: 1, ulcerations; 2, tumours.

Ulcers of the tongue are of manifold nature. Their diagnosis furnishes one of the most difficult chapters of surgery. The ulcers appearing on the tongue are traumatic, syphilitic, carcinomatous, tuberculous, and lupoid; the majority, therefore, are symptomatic. Consequently, in most instances, the diagnostician will be obliged to look for and rely upon distant landmarks.

A tuberculous ulcer is suspected if the individual is of tubercular habitus. It is not true, however, that, as was believed until recently, the patient must be in an advanced state of phthisis. Chvostek has observed a primary, tuberculous ulcer of the tongue. We think of carcinoma if the individual has reached a ripe or advanced age, though it may make its appearance at an earlier period. Syphilis must be thought of in each case, as this disease spares no time of life. It is unnecessary to attempt to obtain an extensive variety of symptoms in each case in order to establish a diagnosis. A case was sent to us for operation with the diagnosis of cancer. The ulceration, which was extensive, involving the tip and part of the left edge of the tongue, was recognised by most of those present as non-malignant, for the simple reason that the ulcer had existed for years without impairing the movements of the tongue, without perceptible swelling of the glands of the neck, and without cachexia. A markedly indurated base, and a single hard and enlarged lymph gland occurring in an individual of advanced age will cause everybody to suspect carcinoma, and that only. A smaller ulcer, near the tip of the tongue, situated opposite a rough, carious tooth, will usually prove to be traumatic.

Upon what landmarks, in the diagnosis of these conditions, can we rely?

1. *Situation.* An ulcer on the dorsum will, as a rule, not be of traumatic nature unless some peculiar trauma has occurred. This can be learned from the history; or some particular local cause may be found, such as a necrotic piece of bone on the hard palate, or some misplaced tooth (situated behind the upper row of teeth), with tartar or carious irregularities upon its surface. An ulcer of the dorsum near the base of the tongue will rarely be syphilitic. These ulcerations are usually placed near the tip, or at the frenum; in other words, anteriorly.

2. *Extent of the ulcer.* An ulcer which spreads far and wide along the floor of the mouth and to the gums will hardly be of traumatic or tuberculous origin; more likely it will be syphilitic, and most probably carcinomatous. Tuberculous ulcers are always small; a very large ulceration will therefore not be tuberculous.

3. *Character of the edges.* Cancer has a very hard edge; the syphilitic ulcer a softer, very sharp border; the tuberculous, a sinuous, dentated, undermined border. Traumatic ulcers possess a hardened, painful edge only after very prolonged irritation.

4. *Character of the base.* The base of a traumatic ulceration, after removing irritating agencies, rapidly becomes a clean, granulating surface. Syphilitic ulcers possess a lardaceous base, which clears up on using potassium iodide. Cancer often contains epithelial pearls, which can be squeezed out by pressure. Tuberculous ulcers have a roughened base, and sometimes show small grayish nodules scattered here and there. Lupus is characterized by small bright-red nodules, composed of papillæ, which bleed readily. They are situated at

the edges of the small ulcers, which are formed by the breaking down of nodules.

Circumscribed NODES in the substance of the tongue are even harder to diagnose than the above conditions. A hard, sharply circumscribed node embedded in the substance of the muscles, and covered by the normal mucous membrane, may be an abscess, a gumma, or a cancerous node. We must not expect to find fluctuation plainly marked. A circumscribed *abscess*—fluctuation, as was just stated, can rarely be felt if the abscess is deeply situated—is very painful. The pain is increased as the tension increases. The mass usually attains the size of an almond. A cancerous nodule is only slightly sensitive; only exceptionally does it become painful. These signs, however, are insufficient; other guides must be found. Age has a decided value: if the individual is young, cancer is excluded. In middle and advanced age any of the mentioned conditions may be present. Complete examination of the patient must be undertaken. Signs of lues elsewhere on the body are in favour of a gumma. If no lues is present, the rapidity with which the node developed is of great importance. An abscess forms in eight to fourteen days; a cancerous node requires a longer time to grow to a corresponding size. A small, round, hard, painless lymphatic gland in the neck points to cancer.

Other rarer tumours appear on the tongue: tubercles, actinomycotic foci, lipomata, or fibromata. Fibromata are always situated superficially. They are flat, small (about a square centimetre in extent), and exist for years. A lipoma is elastic, and likewise requires years to develop. Tubercles are found only in connection with tuberculosis of other organs (lung, bones, or joints), and eventually ulcerate. I refer here to the rare cases of larger nodes, not to the more commonly met with tubercular ulcers of the tongue which were previously discussed. An isolated focus of actinomycosis can be recognised only by exclusion. The first case which was diagnosed at our clinic was recognised in the following way: The individual was young (no carcinoma) and healthy (no tuberculosis or syphilis), and the node had existed some time (no abscess).

As cancer of the tongue very rarely occurs in females, this is to be remembered in diagnosing tumours of the tongue.

If a tumour of the tongue ulcerates, we are dealing with cancer, tuberculosis, or syphilis. By bearing this in mind no great difficulty should be encountered in arriving at a correct diagnosis.

CHAPTER IX

TUMOURS OF THE NECK AND OF THE PAROTID REGION

THE complicated topographical relations in the neck cause some difficulty to beginners in judging the nature of tumours in this region; for the physician well drilled in anatomy, the complexity of structures serves rather as a means of orientation.

Therefore, the data which point to the position, origin, connection, and extension into the deeper parts become more complete and numerous. There are certain definite points by which most tumours of the neck can be recognised. He who possesses the gift of intuition, who recognises and appreciates the symptom complex as a whole, will always be able to make a readier diagnosis than the one who examines and weighs isolated symptoms, regarding them as disjointed pieces of a complete structure. Dieffenbach called this perception by means of inspection *autopsy*, and gave Rust great credit for so assiduously exercising his pupils in this branch. Therefore, it is advisable to study these tumours in the grouping in which they are wont to be encountered in practice, and not according to the pathological divisions to which they belong.

Let us consider, first of all, the congenital tumours of the neck. Most striking, because of the whole train of symptoms, is the congenital cystoid of the neck, HYGROMA CYSTICUM colli congenitum. According to the researches of Köster, it is an ectasis of the lymph vessels. It consists of a tumour composed of fluctuating lobes, which are more or less connected with each other. As a rule, it first makes its appearance in the submaxillary region, but soon, by its rapid growth, extends upward over the cheek and downward over the neck. The most serious symptoms of compression of various organs arise, when the tumour penetrates into the deeper layers. It follows that children afflicted with this disease rapidly succumb, during the first months of their existence, as the result of impaired nutrition; but cases are met with in which pressure on the air-passages causes a much earlier fatal termination. The patients very rarely reach an adult age. As the tumour, in its rapid growth, penetrates everywhere, between and about the organs, its extirpation can be thought of only in the fewest instances. The cystic swellings are situated both in the superficial and deeper tissues, therefore some will be plainly fluctuating and covered by a thinned-out layer of skin, while others, more deeply placed, will not show fluctuation as unmistakably. In some cases the skin is thickened by elephantiasis. This form of swelling is sufficiently characterized by its appearance at birth, its rapid growth, and the multiple number of ectases.

Simple cysts also appear congenitally. They are related to the developmental processes of the organs in the neck. We know that they originate from a branchial cleft obliterated at both ends. They have therefore been called *Branchiogenetic Cysts.* Recently Rabl has shown that the cysts do not originate from the branchial canals which lie between the arches, but from a passage which extends from the second branchial cleft to the sinus cervicalis. Rabl calls this canal branchial, therefore we may continue to employ the name of branchial cyst with, however, a new significance. If the cleft has closed toward the inside, but not toward the outside, an external *Branchiogenetic Fistula* results; if the obliteration is complete externally but not internally, we speak of an internal fistula; if no closure has taken place, the fistula is complete. The external opening is situated directly above the sterno-clavicular articulation; the internal is at the pillar of the fauces.

Clinically, branchiogenetic cysts appear as rounded, elastic, fluctuating tumours, which are found in any region of the neck, from the lobe of the ear to the jugulum. They may be attached to the lower jaw, to the hyoid bone, or to the styloid process, but most frequently to the sheath of the great vessels. Their contents is pulpy, sometimes oily, in consistence. We are indebted to Hochenegg for a point in diagnosis which may readily be applied. It consists in laying an ice-bag upon the tumour; if the contents is pulpy or oily it congeals, and the tumour grows solid. Sublingual dermoids stand in close relation to the above-mentioned more laterally placed tumours.

The *Cartilaginous Skintags* found in the neck, and first described by Weinlecher, owe their origin likewise to embryonal processes of development. As a rule, they are small, symmetrically placed appendages, which inclose a bit of cartilage in their interior—probably a remnant of a branchial arch.

In the third place, goitres occur in the new-born just as in adults. Their diagnosis will be discussed later.

Finally, there remains to be described a tumour which appears in the first few days after birth, causing great perplexity to the beginner, as I saw in two cases which came to my notice. An elongated swelling, firm, tense, and sharply circumscribed, embraces the whole, or the greater part, of the sterno-mastoid. Whether it is painful or not can not be determined in the new-born. In some cases the child screams whenever the tumour is lightly touched, thus giving the impression of an inflammatory process.

The acuteness of the pain passes away in a few days. It is nothing more than a MYOSITIS of the sterno-cleido-mastoid, due to difficult labour. Breech delivery, in which the aftercoming head has to be pulled out with great force, usually produces the condition. The head of the infant is displaced toward the sound side. *This must be kept in mind*, as we are more apt to expect the inflamed muscles to be contracted (similar to the inflamed psoas) and the head bent toward the affected side.

Quite as confusing to the beginner is the appearance of one or more hard swellings in the substance of the sterno-mastoid of a new-born child. Dieffenbach, not so long ago, in showing the first few cases which came under his observation, described them as something new, and not previously observed. These are RUPTURE of the sterno-mastoid, due to difficult labor. As a rule, the parents bring the infant to be examined several days after birth, and the physician is apt to regard it as an adenitis. Dieffenbach, as early as 1830, made the following clever observation: "The case has a striking resemblance to newly healed fractures of the clavicle. Although the fracture passes unnoticed by the parents, the callus attracts attention. They seek the physician in order to find out whether the swelling is not an enlarged gland." This shows that even in the bygone days in which Dieffenbach's report appeared, in Rust's Handbook, parents held the same view of surgery as the parents of to-day.

The diagnosis of the cervical swellings seen in adults will be treated without strict classification, presenting them as a series of pictures.

GOITRES head the list of tumours of the neck because of their frequency (at least in certain countries), their size, their complicated pathological anatomy, and the important secondary symptoms caused by them.

The practitioner inquires, Are we dealing with a tumour of the thyroid gland, and what is its nature?

The tumour is connected with the thyroid: (1) If it lies beneath the muscles which cover the gland. As soon as the tumour is somewhat larger, and extends laterally, it is covered chiefly by the sterno-mastoid muscle, which can be raised away from the tumour, by grasping its upper or lower insertion. (2) If the tumour is of granular structure, or is embedded in the granular structure of the thyroid, which closely surrounds it, and is directly continuous with the gland. (3) If it rises and falls during the act of deglutition.

San Francisco, Cal.

Tumours of the thyroid gland may be classified as follows:

(*a*) *Parenchymatous goitre*, if the whole growth is composed of small granules of approximately equal size.

(*b*) *Cystic goitre*, if we are able to distinguish rounded, sharply circumscribed, elastic, and fluctuating nodules in the granular substance of the tumour.

(*c*) *Hyaline degeneration* of one or more nodules, if a rounded, circumscribed, elastic, soft nodule is prominent, but in spite of its superficial location gives no signs of fluctuation. To distinguish between this degenerative variety and a cyst is almost impossible, if the nodule in question is deeply embedded in the mass, and therefore does not give unmistakable signs of fluctuation.

(*d*) *Fibrous goitre*, if larger, very hard lobules are present, with small granular structure at their periphery. This is due to a hyperplasia of the follicles at the periphery, which goes hand in hand with the formation and sclerosis of the interstitial connective tissue.

(*e*) *Vascular goitre*, if in addition to distended superficial veins, the tumour can be reduced on pressure, but immediately regains its former size when the pressure is removed. If there are numerous arteries which pulsate and cause a bruit, it follows that the arteries are dilated and increased in number (struma aneurysmatica).

Malignant tumours not rarely develop in a thyroid gland, which has previously shown signs of enlargement. The tumour in question may be a sarcoma or a carcinoma. The striking rapidity of growth within a recent period is the most noticeable symptom of malignancy. A whole series of other symptoms may be added to this. Pain is felt in remote parts; as a rule, it is situated in the occiput or shoulder, rarely in the arm. The gland feels more homogeneous: a single hard mass. Malignancy can be recognised early by involvement of neighbouring structures (muscles, esophagus); by symptoms referable to the sympathetic system (pupillary changes, position of the bulb); and by marked enlargement of the veins situated over the sternum (due to compression or obliteration of the veins of the neck).

Later in the course of the disease metastases give characteristic signs (spontaneous fractures of bones, symptoms of tumour at the base of the skull).

A patient suffering from malignant tumour of the thyroid could not swallow, but an esophageal bougie passed with ease. How was this possible? The autopsy explained the condition, though it had been previously suspected. The pharynx was almost completely surrounded by the growth, therefore rigid and incapable of contraction.

A youthful subject had a tumour of the neck, which, seen from a distance, appeared to be a goitre. The tumour extended laterally, on both sides of the neck, behind the sterno-mastoid. Closer examination showed that the carotid of the left side was *in front* of the tumour and pulsated *visibly;* on the right side pulsation could not be seen, but could be *felt*. Goitres crowd the carotid back—that is, lie in front of the artery. Here the tumour, which fluctuated, was posterior to the vessels. This apparent incongruity directed attention to a previously unnoticed rigidity of the neck. Diagnosis: Bilateral cold abscess, due to tuberculosis of the cervical vertebræ. Incision evacuated pus.

LYMPHATIC TUMOURS of the neck are of very frequent occurrence, forming the so-called chainlike tumours (*Kettengeschwulst*) which extend down the neck. These swellings correspond, in their anatomical situation, to the position of the various groups of lymph glands: the submaxillary glands, the glands situated in the carotid triangle between sterno-mastoid and the larynx, and finally the glands placed behind the sterno-mastoid, about the jugular vein, extending to the supraclavicular fossa. Frequently all three groups, sometimes bilaterally, are swollen, so as to form multilobular masses composed of very many sharply circumscribed nodes. In other cases a well-marked swelling may be found, for instance, in the submaxillary region; while the palpating finger detects only the beginning of this chainlike enlargement along the inner edge of the sterno-mastoid the supraclavicular fossa of the same or the opposite In the great majority of cases this is the picture ented by scrofula, a disease of childhood, prevalent pecially among the poorer classes. As careful physi- ians we should not fail to examine the nose, mouth, and pharynx of the patient, in order to determine whether a carious tooth, an ulcer, or a catarrh may not act as a local cause of infection and secondarily produce a swelling of the glands.

If the individual is healthy and not of tubercular habitus (we must assume that the tubercle bacillus is unable to infect every person) one or more lymph glands may enlarge because of some ulcerative process on the head; but the swollen glands resolve as the infection subsides. If an extensive invasion of pus cocci has taken place, an acute suppurative lymphadenitis will result. Tubercular infection of the glands leads to chronic adenitis, and, as a rule, to cheesy degeneration or suppuration of the glands. After breaking down, the characteristic tubercular ulcerations result, and are marked by violet-coloured, deeply undermined and sharply cut edges. When these heal they leave typical multiple, radiating scars, with raised borders, sometimes bounded by very small tags of skin. Cheesy degeneration and softening, in the shape of a cold abscess, is distinctive of tubercular adenitis.

In each case a peripheral point of origin of the trouble must be suspected and sought for, just as in the case of a syphilitic bubo we examine the genital region for a chancre. During this examination, it is necessary to keep in mind the peripheral regions drained by each set of lymphatic glands in the neck.

Multiple *leukæmic* enlargements of the lymph glands appear in the neck. The swellings are hard, separately movable, and never attached to the skin. They rarely exceed a walnut in size, never soften, and are painless. Similar swellings are found in the axilla and groin.

That peculiar disease of the lymphatic glands which was considered MALIGNANT LYMPHOMA by Billroth is now commonly called lymphosarcoma. A whole group of glands enlarges, the individual glands remaining discrete, freely movable, and of varying consistence.

As a rule, the swelling first appears in the submaxillary region, then along the sterno-mastoid, and in the supraclavicular fossa. The swelling can attain enormous proportions. In the course of a short time metastases are noticed in other parts of the body, and the patient succumbs within two years, whether the glands are extirpated or not. If the above-mentioned picture is encountered, and no local or general cause for the swelling can be found, the diagnosis of malignant lymphoma may be made by exclusion.

The name of lymphoma, as applied to non-inflammatory swellings of the lymph nodes, includes lymphoma simplex, lymphoma scrofulosum, and lymphoma malignum.

Lymphatic tumours situated laterally and high in the neck, and showing a marked tendency to infiltrate, may give the impression of a sarcoma, but sometimes are secondary carcinomatous glands due to a hidden and deep-seated primary focus—for instance, in the sinus pyriformis.

Sarcomata of the spinal column may grow either laterally or directly forward, crowding the organs before them, thus producing dysphagia and dyspnœa.

They are a *noli me tangere*, and their apparent mobility is deceptive, for the tumour may be attached by a slender pedicle, and then appear to be mobile. The nature of this very rare growth must be determined in each case according to general principles.

As in other parts of the body, *single* tumours of the neck may be systematically classified. For example, they may be either solid or fluctuating. If the tumour fluctuates, or is unmistakably elastic, it contains fluid, which may be cyst fluid, blood, or pus. If the contents is blood, the tumour is compressible, and is an angioma, or a blood cyst, or an aneurism.

An *angioma* is not sharply circumscribed, is of soft consistence, and has cutaneous vessels, which shine through the skin with a bluish tinge, coursing over its surface. It can not be mistaken for any other condition. A *blood cyst*, which can be emptied, is a rounded, readily palpable tumour. An *aneurism* pulsates. If a tumour is not compressible, and does not pulsate, its contents are neither venous nor arterial blood. Such a swelling must contain pus, turbid cyst fluid, or clear fluid. The latter is at once characterized by its translucency, and the tumour is recognised as a cyst. Doubt arises when the tumour does not contain blood and is not translucent. Such a tumour may be a sebaceous cyst, or an enlarged bursa, or a COLD ABSCESS. (In an acute abscess the inflammatory symptoms are so evident that it could not be confused with these other conditions.) Only deeply placed cold abscesses, which are rounded, tense, and sharply circumscribed, due to a broken-down lymph gland, are included here. The following points should be looked for:

1. Slow, almost painless suppuration takes place

only in individuals of lymphatic habitus. 2. As a rule, more careful examination will show that one or more of the neighbouring glands are enlarged. 3. The swelling is of some size from the outset, for lymph glands begin by enlarging before they suppurate. Cysts, however, can already be distinctly felt when barely the size of a pea. 4. In broken-down glands spots of harder consistence can be felt here and there. 5. The shape is not strictly rounded.

A cystic bursa is distinguished by its site—either in front of the thyroid cartilage or symmetrically placed beneath the hyoid bone—and by its immobility. The bursa under the hyoid bone, especially, is differentiated from a cyst by its flattened shape. The bursa in front of the thyroid cartilage is characterized by its rounded form, soft consistence, and, if it contains rice bodies, by the friction crepitus they produce.

How are we to decide between an inflamed bursa situated in front of the thyroid cartilage, and a suppurating lymph gland in this situation? Such a dilemma can arise only if the lymph gland has broken down very slowly, and has not grown painful until ready to break through the skin. The previous existence of a soft swelling must be taken for granted, as the bursa would become inflamed only after an hygroma had persisted for some time. The following points will aid: 1. The individual. Slow, painless breaking down of a lymphatic gland occurs only in people of tubercular habitus; bursæ are found in muscular subjects. 2. Inflammation of the bursa is marked by sudden occurrence of severe throbbing pain, with œdema and redness of the skin. The suppurating tubercular gland causes a circumscribed reddening of the skin, is gradual in its onset, and is not accompanied by the same throbbing pain. The fever noted in the suppurative bursitis is absent in tubercular adenitis.

Large, flabby, cold abscesses of the neck occur only in connection with bone trouble, especially in disease of the spine. In such cases the symptoms of the spinal trouble are present.

The tumours which contain blood communicate directly with a blood-vessel, and consequently empty on pressure. They are either blood cysts—which communicate with veins, large varices—or ANEURISMS. If the volume of the tumour increases on compression of the efferent vein, the connection with the vein is demonstrated. Aneurisms, on the other hand, pulsate. We may therefore say that a compressible tumour contains blood, and that a pulsating tumour contains arterial blood; consequently it is an aneurism—either a true or a false one. In the case of an aneurism, we have only to determine the vessel from which it springs in order to complete the differential diagnosis.

A cavernous angioma, in addition to its compressibility, is characterized by other external signs. Its surface is flat and irregular, yet the swelling is not truly fluctuating. Unfortunately, while theoretical proof is easy, the practical proof is hard to obtain. To show that a tumour of the neck is compressible is sometimes a difficult feat.

We have taken for granted that pulsation may be relied upon as an entirely unequivocal sign, which justifies the diagnosis of an aneurism if the tumour can be emptied. We must modify this statement by adding, that in practice this simple and convincing basis can frequently not be applied, for the reason that at the very outset of our examination we will be greatly embarrassed, and unable to distinguish whether the tumour can be emptied—*cum grano salis*, we may say that it is only compressible—or in fact whether it pulsates. It is not easy to empty an aneurism, and, further, in carotid aneurism we refrain from all heavy pressure or kneading of the tumour for diagnostic purposes. Ferguson employed manipulation of the sac to effect a cure. Yet a still living and highly successful surgeon, Professor Esmarch, of Kiel, was unfortunate enough to have a patient become hemiplegic and die as a result of kneading, the autopsy showing that widespread peripheral thrombosis had resulted. The patient's death, therefore, was directly due to digital examination. On the other hand, the tumour under consideration may show *transmitted* pulsation. An abscess, which resembles an aneurism in shape and location, may transmit pulsation, but repeated examinations will show certain well-marked differences.

We are guided by the fact (1) that when the neck is held properly the swelling shows no lateral pulsation, and that when it is lifted away from the artery all pulsation ceases; (2) that at each diastole the swelling is raised, but not enlarged; (3) that no bruit is heard.

All the signs and symptoms of an aneurism may be present—pulsation, diastolic increase in size, and bruit—and yet the tumour in question not be an aneurism. It may be a very vascular neoplasm; differentiation is then very difficult. It is true that such a neoplasm can not be emptied by pressure, but, as it is soft and more or less compressible, the symptoms are not convincing.

Observation of the pulse is a more trustworthy guide. In a large aneurism the pulse in the peripheral branches of the vessels is retarded by a small fraction of a second; a neoplasm situated close to the carotid or subclavian can not cause this symptom. In addition, knowledge of the manner in which the tumour has developed affords valuable confirmatory evidence. A neoplasm develops rapidly, an aneurism slowly. The patient may tell us that at first the tumour was a hard nodule; that it began to soften later, and that pulsation grew more and more distinct. This harmonizes with the history of a tumour, for an aneurism is soft and pulsates from the outset. If a pulsating tumour rises and falls during deglutition aneurism is at once excluded. Attention was first called to this fact by Astley Cooper.

In some cases the question arises from which of the large vessels of the neck the aneurism is derived. The importance of this query is evident, even if distal ligature is determined upon. If the aneurism is placed at the bifurcation of the carotid, we may be in doubt whether the external or internal carotid is involved, but the question is of no importance, as in either case the main trunk would be ligated. If the aneurism, however, is situated at the origin of the carotid, it is important to be certain whether the dilatation is of the carotid, subclavian, or innominate arteries. Mistakes have here been made by experienced surgeons. The most important symptom is the retarded pulse in the peripheral twigs of that artery which is the seat of the aneurism.

The point of origin of tumours of the parotid region can deceive the most experienced diagnostician. This holds true not only of tumours, but also of inflammatory swellings. Endemic PAROTITIS gives a characteristic picture, recognised even by the laity, as is well shown by the peculiar popular names given to it. The names of "goat peter" (*Ziegenpeter*), *Bauernwetzel*, or mumps, do not show the histology or the site of the disease; but "booby sickness" (*Tölpelkrankheit*) is more enlightening, as it offers but one explanation—that the appearance of the patient is like that of a lout. The designation of *Wochentölpel* (literally, booby sickness of a week's duration) sheds light not only on the clinical features, but also upon the duration of the disease. The rapid and extensive swelling in the parotid region causes the neck to become broader than the head, especially if a symmetrical enlargement of both sides takes place. The patient experiences difficulty of speech, and suffers from dribbling of saliva, for swallowing becomes painful. He is unable to turn his head; his neck is misshapen; his entire appearance is loutish—*inde nomen.* In a case of this kind an error in diagnosis is scarcely possible, for the lymphatic glands never swell to this size in the course of a few days. If we meet with the disease in its early stage, we rely on the following symptoms: the patient has pain behind the ramus of the jaw and below the ear, ushered in by sudden fever, or even by a chill. A swelling appears in the region mentioned, and the hollow normally found anterior to the sterno-mastoid is replaced by a prominence. This swelling is painful on pressure, and not circumscribed. The skin covering it is not reddened, but paler and more waxy than normal. The patient anxiously avoids opening his mouth, because this act would compress the parotid, and therefore cause great pain. Most patients are annoyed by tinnitus aurium. The affected side of the mouth is either drier, because the salivary secretion is inhibited, or there is increased salivation. The saliva sometimes is turbid and fetid. The only other condition which could bring about this symptom-complex is an inflammation of the lymphatic glands lying beneath the parotid fascia. As these glands are numerous, and many of them are embedded in the parotid gland itself, it appears reasonable that this inflammation could cause a sympathetic disturbance of the parotid—abnormality of secretion. Therefore, the diagnosis between these two conditions would appear very difficult were it not that the physician is guided from the very start by the fact that the tissue of the entire parotid is uniformly nodular and painful to the touch. At a later stage, the rapid increase of the whole swelling is of such proportions as to exclude its origin from the small lymphatic glands. If the swelling disappears without suppurating, the case is even more clearly demonstrated, for only deep-seated, suppurating glands can cause a swelling of large proportions. If the disease is endemic, no doubt as to the nature of the trouble can arise; likewise, if the swelling of the parotid is followed by swelling of the testicle.

But there is another variety of parotid swellings which is still almost invariably unrecognised. These are the so-called salivary tumours, or, as they are more properly called by v. Bruns, CYSTS OF THE SALIVARY DUCT. They are due to local distention of the salivary duct by a collection of saliva in the sac. As a rule, they do not reach large dimensions. If no inflammatory symptoms are present the swelling is most often mistaken for a cyst, for it is sharply circumscribed, fluctuates, and, in many cases, tense. In a scrofulous individual it may be confounded with a cold abscess, or, if more violent inflammatory symptoms are not wanting, with an acute abscess. If, labouring under this mistaken diagnosis, the swelling is incised, the resulting fistula is hard to close; this error should therefore be avoided.

If the tumour can be partially emptied, this symptom, alone, demands examination of the inner end of Stenson's duct by probing from within the mouth. Further observation is then required, and special attention should be directed to the tumour on chewing, speaking, or on partaking of salty or acid food, for the swelling may increase in size with the additional secretion of saliva. In doubtful cases an exploratory puncture, made from within the mouth, should settle the matter.

The following case was of interest: A man, about forty years of age, came to the clinic complaining of a flattened, rounded tumour, the size of a half walnut, situated in the parotid region. The tumour was sharply circumscribed and fluctuating. The skin over it was normal. The patient was healthy: therefore no cold abscess. Consequently, a sebaceous cyst was the hasty diagnosis of the students. I, however, showed that the tumour was immovably fixed, and that the skin did not show the vascular network which is invariably present in sebaceous cysts of this size occurring on the face. I diagnosed a salivary cyst, and puncture showed salivary secretion.

Not infrequently *salivary fistulæ* occur in the parotid region. The location of the fistula will usually determine whether the tract leads down to the duct or to a lobe of the gland. If its site does not definitely settle this point, the amount of saliva discharged will do so. A *fistula* of *the duct* alone can discharge *large* quantities of saliva after eating, because it receives the secretion of the whole gland.

Solid *Tumours* of the parotid often offer insurmountable difficulties to diagnosis. Naturally, only those tumours which extend deeply into the parotid fossa, and are not readily movable, are here referred to. A careful history is of the greatest assistance. A tumour which begins as a small, protruding, movable nodule, and grows fixed only after penetrating into the deeper parts, did not originate from the parotid gland. A tumour which at first forms a flat and diffuse elevation in the parotid region, but later develops into prominent knobs, must have started from the parotid. This really is all that can be given as a general characteristic.

CHAPTER X

DYSPHAGIA—STENOSIS OF THE ESOPHAGUS

A PHYSICIAN of Amsterdam (van Geuns) more than one hundred years ago divided dysphagia into two varieties. He distinguished between difficulty of deglutition and difficulty of transglutition. In the first, the patient can begin the act of swallowing, but the bolus sticks fast in the esophagus; in the second, the solid or fluid meets with hindrance somewhere between the lips and the gullet. The bolus can not reach the esophagus, but, if it reaches this, it can enter the stomach without further hindrance. This division is of practical value, for the diseases which produce difficulty in the chewing, formation, and transportation of the bolus differ from those which are marked by an obstacle situated in the esophagus itself. In the former, paralysis is usually the cause, as chewing and turning the bolus, and then transporting it to the esophagus, is chiefly a muscular act. The latter, as a rule, is caused by strictures; for Mikulicz, by means of esophagoscopy, practised from above, and Maydl, by endoscopy through a gastric fistula, from below, showed that the normal esophagus is a wide-open and gaping tube into which the object to be swallowed need only be introduced. In order to appreciate the value of the symptoms, it is sufficient to be cognizant of these facts. Swallowing presupposes not only patency of the whole passage, from the lips to the cardia, but also unimpaired muscular action. Therefore the main inquiry will be directed to find out whether a stricture is present or not. Inspection of the mouth and pharynx, examination of the esophagus by bougies, will always enable us to detect a stricture. It will become merely a question of detail and of technic. If stricture can be excluded, either muscular paresis or some obstruction of the upper passages, which are open to inspection, is the cause of the trouble.

Before going further, it is well to emphasize the fact that a stricture of the lower end of the esophagus may exist, and that the physicians may regard it as a case of gastric trouble. Such a barely conceivable error results from omitting the examination by bougies.

Trousseau mentions the case of a woman who was treated for many years for supposed gastric troubles, in consequence of which she had greatly emaciated. By examination of the esophagus with bougies a stricture was detected, and the patient's health restored in the course of a few weeks.

Steiger recounts an analogous case. A gentleman suffered from frequent vomiting of food and mucus, retching, and what was supposed to be gastralgia. The patient's age, emaciation, and cachexia led to the diagnosis of cancer of the stomach, as he always asserted that his ability to swallow was unimpaired. The bougie, however, detected a considerable narrowing of the esophagus at the cardia, which later proved carcinomatous.

Examination of the esophagus by the bougie is the most important diagnostic aid, but as it is not always submitted to by the patient, those signs which point to esophageal disease had best be mentioned. If auscultation of the esophagus were unimpeachable, it might be of value in making a provisional diagnosis.

Except in such unusual cases as those mentioned above, it is, as a rule, possible to make the diagnosis of stricture from the facts gathered from the patient. A positive cause may be known: as, for instance, the swallowing and impaction of a foreign body, or the swallowing of some caustic substance. In such cases the diagnosis is confirmed by seeing the patient regurgitate food, or by his locating a definite spot in the neck at which the food remains fast. The bougie is then used at once, as in foreign bodies, or it will have to be used later, as in strictures; never in cases of burns by caustics, until some time has elapsed, as the injured spots should not be sounded before cicatrization has taken place. This stage can be recognised by the fact that bloody pus is no longer vomited or regurgitated.

Let us take a case in which dysphagia has arisen without known cause, and where, consequently, immediate examination by bougie is unnecessary. The patient tells us that he has noticed for some time that solid food stuck fast in his esophagus. As a rule, he will state at what site the arrest occurs, and will add that a swallow of liquid softens and carries down the food. When the patient is no longer able to do this, he is reduced to swallowing liquids, and these only in small quantities. Half a tumbler of water is immediately thrown up with great violence, but a spoonful is well borne. If the patient has not attempted to keep up his strength by taking very concentrated food, such as milk and eggs, he is emaciated when first seen. If the individual is of advanced age, if examination of neck, thorax, and abdomen shows no tumour, we are usually correct in diagnosing CARCINOMA OF THE ESOPHAGUS. This assumption is strengthened by the presence of other symptoms, such as several hard, enlarged, and painless lymphatic glands in the supraclavicular region. Occurrence of spontaneous pain at the seat of stricture, occasional traces of blood in the regurgitated food, and, at a later stage, fetid odour from the mouth, all make the diagnosis more probable. If carcinomatous cells are found in the vomitus the diagnosis is assured. Carcinoma is by no means excluded if the patient states that he has temporary periods of improvement, for parts of the growth may break down, be cast off, and the passage become free. In a short time renewed growth again obstructs the lumen, and this process may be repeated several times. On the whole, however, the disability continually increases with the usual rapidity of carcinomatous growths; therefore the duration does not exceed more than a few months. I have never, as yet, been deceived in the diagnosis of carcinoma by thus interpreting the symptoms.

It must be emphasized that only in the rarest instances can carcinoma of the esophagus be made plainly evident. Swelling of the glands is rare; fragments of the growth, available for microscopical examination, are never encountered. And yet the diagnosis is made with a probability bordering on certainty. Having heard the patient's history, and having demonstrated a stricture by means of bougies, we reason as follows: The patient is of the age at which carcinoma of the esophagus commonly occurs. The stricture has no known cause—in other words, it is spontaneous. Its course has been so rapidly progressive that carcinoma alone could account for it; besides, the patient gives no evidence of other disease. Of course, one other complaint must be ruled out: this is compression of the esophagus by a tumour.

Carcinoma of the esophagus can develop at various parts of the tube. It is most often found at the lower end; or, more accurately, it is found oftenest at any part of the esophagus which is below the upper border of the thorax.

If the obstacle is situated at the level of the cricoid cartilage, we must bear in mind those rare cases of enlargement of the lamina of the cricoid which are sometimes met with. It has been noticed they are attended by very great dysphagia.

Spontaneous stricture of the esophagus may be due to syphilis; but, compared with carcinoma, this occurrence is extremely uncommon.

The causes of stricture are evident if a *tumour of the neck* presses on the esophagus. It is well to remember that goitres in women may *periodically* cause difficulty of deglutition of a minor degree when the tumour undergoes swelling and congestion at the menstrual period.

Pressure stenosis of the esophagus, due to *tumours of the thorax*, is much more difficult to diagnose. Here the methods of diaguosis at the command of internal medicine must be resorted to.

The opposite of stricture is dilatation; sometimes this follows and is the result of stricture. That portion of the tube situated above the stenosis dilates to a moderate degree throughout its whole length.

In addition to this condition, there is a primary dilatation known as *Ectasis*. The esophagus dilates, and becomes a wide, tortuous tube, with an accompanying thickening or thinning of its walls bearing a faint resemblance to the intestine. This is followed by accumulation of the ingesta in the tube, and by the decomposition of the retained masses. The food is vomited; eructation of foul-smelling gases annoy the patient, and an unpleasant feeling of pressure in the chest may follow each meal. The etiology of such conditions is not clearly understood. It appears that most cases are due to paralysis. In former days surgeons spoke of this condition as *Atonia œsophagi*. One of the best-known medical writers of the sixteenth century (Felix Platter) suffered from this disease for several years. Willis mentions one case in which a man continued to live for sixteen years in this condition by shoving his food down the esophagus with a small sponge attached to a whalebone handle. The disease, if at all marked, can be recognised by means of the bougie. A specimen preserved in the museum at Vienna will admit a man's arm. In such an esophagus the sound can make wide excursions. The older authors describe, in connection with this, atonia esophagi—*gulæ imbecillitas* was mentioned by Galen—an extremely anæmic condition of the pharyngeal mucous membrane. Such findings, if a trauma had preceded—a concussion of the thorax, for instance—would support the assumption of a *paralytic origin* of the trouble.

Circumscribed local dilatations of the esophagus are known as Diverticula. The dilatation may be cylindrical, spindle-shaped, or saclike in form. The last variety, in particular (in which the diverticulum has grown to be a pouch placed next to the esophagus and opening into it), is of the greatest surgical interest. It may be due to some ulceration, or to the traction of cicatrizing lymphatic glands situated in the neighbourhood of the esophagus. In addition, there are diverticula which are the remains of fœtal structures. Esophageal diverticula, placed at the apex of the bifurcation of the trachea, are representatives of this class. When the dilatation has reached a certain size it causes disability. The food passes partly into the stomach, partly into the diverticulum, which may be situated in the neck or near the stomach. With the increased fulness of the pouch the patient experiences distinct sensations at the site of the diverticulum. In the neck a visible tumour appears, which to the palpating finger imparts the same peculiar sensation experienced on examining the crop of a bird. In this given case the patient can empty the pouch by squeezing or kneading it. In other cases, the patient can entirely or partially occlude the opening of the diverticulum by holding the neck or chest in certain positions, the food then reaching the stomach unhindered. The bougie meets with the same fate as the food, sometimes reaching the stomach without trouble, at other times becoming engaged in the pouch, and abruptly arrested. In some of these patients a peculiar phenomenon can be observed: the food returns to the mouth shortly after a meal, without vomiting or retching, just as it does in the ruminants. A well-known medical scientist, who was also a shining light in Parliament, called an opposing political party ruminants. He escaped being called to order only by stating that *cud-chewing* was a *human infirmity*. We might as well state here that cud-chewing can occur independent of a diverticulum.

Weinlecher makes the important statement that the opening of the pouch gradually changes its position during its development. If the patient is under observation for a long time and is frequently examined, it will be noticed that the place at which the bougie meets with obstruction by degrees assumes a different level.

A gradually increasing degree of stenosis, arising without any known primary cause, may be due to an esophageal polyp. The esophageal bougie discovers nothing more distinctive than resistance to its progress; therefore the diagnosis of **Polyp**, in distinction from other neoplasms, must be made by signs characteristic of the condition. These distinctive symptoms are due to the site and the mobility of the growth. It develops behind the larynx; consequently hoarseness is often noticed at the beginning of the trouble, and at a later stage the laryngoscope may detect some change in the position of the structures inside the larynx. The polyp becomes more movable as it increases in size. In addition to obstruction in swallowing, it causes retching, not only during deglutition, but also at other times. Severe attacks of choking, which occur now and then when the polyp is forced up by the retching and comes to lie over the aditus ad laryngem, are characteristic of this condition. Death from asphyxia has been known to result. All doubt is removed if the polyp becomes visible, or if the bougie, as sometimes happens, detects the mobility of the tumour.

Just as a spasmodic stricture of the urethra is met with, a similar *spasmodic* narrowing of the esophagus without doubt occurs. Those cases in which there is a fissure in the mucous membrane of the tube are readily explained. When a particle of food passes the sensitive spot a reflex spasm may take place. Some surgeons state that in such cases they noticed the distinct sensation of the bougie being grasped by the esophagus. We are reminded by the symptoms of cases of fissura ani, in which likewise a reflex spasm of the sphincter is present; also of vaginismus, in which a reflex spasm of the sphincter vaginæ is found. For this reason the disease has been called ESOPHAGISMUS.

If other spasms of the esophagus occur, it is evident that the stenosis caused by them must be characterized by *an alternation between complete permeability and complete stenosis.* When these symptoms are noticed no uncertainty should be entertained; but, as these strictures are found in hysterical and hypochondriacal patients, their symptomatology varies greatly. Of the utmost importance is the observation that hysterical patients give very *many symptoms.* Sometimes such a woman will be unable to swallow hot, at other times cold, food; sometimes solids, at other times fluids, at times nothing passes down. She is afraid that her gullet is "growing together," and that she will have to die of starvation; or she fears eating anything lest immediate strangulation result. The experienced surgeon will know, after a few minutes of listening, with what condition he has to deal. In all cases, examination with the bougie remains as a positive means of diagnosis. As a rule, however, any treatment directed against the hysteria (often psychical treatment) is sufficient to relieve the condition without examination.

Boyer mentions the case of a woman who swallowed solid food only with greatest fear and trembling. He cured her by taking his meals with her, twice daily, for a month, and convincing her of the idleness of her fears. Sauvages treated a similar case with a daily cold bath, combined with milk diet. *Alii aliter.* Older authors mention that some patients are able to swallow large quantities of fluids rapidly, and at one gulp, but never slowly and gradually. The same can be seen to-day, without the least suspicion of hypochondria, at the drinking-bouts of the so-called Foxes (a student society).

Next to carcinoma of the esophagus, no more pitiable condition is met with than RETRO-ESOPHAGEAL

ABSCESS. It may run a chronic course as the result of caries of the vertebræ. The primary trouble is then evident (the kyphotic prominence caused by one or more spinous processes), and can be readily demonstrated. The dysphagia increases gradually. When the bougie is introduced it reaches a sensitive spot, which is the seat of the obstruction. Such an abscess may also be due to perforation of a foreign body. The literature of the subject contains a number of sad cases, in which an insignificant foreign body penetrated into the retroesophageal connective tissue, suppurated, and caused fatal mediastinitis, pleuritis, or pericarditis. Finally, such an abscess may arise idiopathically and run an acute course. Although the symptoms of stricture of the esophagus are indispensable in order to make the diagnosis, the above-mentioned severe complications occupy the foreground of the picture. They may occur in consequence of inflammatory stricture (analogous to periurethral abscess), or as a complication of carcinoma of the esophagus by spontaneous perforation, or as the result of accidents in probing, in spite of the greatest skill and care. The perforation may take place into the mediastinum, pleura, lung, bronchus, or into a large blood-vessel; in the latter case death results almost instantaneously. In the other cases the symptoms are always very grave. If perforation into the air-passages has taken place, we would expect subcutaneous emphysema as a direct result. But this is by no means necessary, for the tissues are usually so glued together, previous to perforation, that the air is unable to make its way into the connective-tissue planes. Rupture due to probing is more often followed by emphysema. Coughing and spitting up of substances just swallowed is characteristic. If the coughing-fit occurs immediately upon swallowing, we must suspect a short and wide communication; if the cough appears somewhat later, the communicating passage is more apt to be long, tortuous, and narrow. If perforation into the lung has taken place, pneumonia will soon appear, while perforation into the pleura or mediastinum is rapidly followed by pleurisy or mediastinitis.

The highest grade of stenosis is complete closure of the passage. Cicatricial strictures and pressure from extraneous tumours cause a relatively impervious stricture—i. e., one which will not permit the passage of the smallest instruments.

There are cases in which water can pass only drop by drop. It is well to bear in mind that in such cases, by changing the position of the patient, we may at times succeed in passing a small bougie. In new-born infants attention must be fixed in another direction. There are cases of complete *congenital closure* of the esophagus—i. e., a blind termination. Examination with an elastic catheter will disclose at once that the child is sure to die unless a gastrostomy is performed.

In the first months of infancy only one form of interference with transglutition is met with, and even that is very exceptional. For that very reason I desire to draw it to your attention, because we are not likely to be on the lookout for it. The trouble referred to is RETROPHARYNGEAL ABSCESS, which has been mentioned in a previous chapter. The child holds its head rigid and immovable. Nasal catarrh is present, the palate is swollen, and a tender mass appears on the side of the neck posterior to the ramus of the jaw. In addition, difficulty in deglutition is met with, sometimes to such a degree that all food which the child attempts to swallow immediately regurgitates through the mouth and nose. Laboured breathing and attacks of suffocation also occur. If these symptoms are noticed, examination of the posterior pharyngeal wall by the finger is called for. To omit this would be an act of grossest carelessness. In larger children, more careful examination will show whether the cervical vertebræ have undergone any change which would indicate caries of the vertebral bodies, or at least permit us to determine whether some part of the cervical spine is sensitive to pressure.

A disturbance of transglutition, confined almost entirely to childhood, occurs as a sequel to DIPHTHERITIA. The case may have been one of simple diphtheria, or a diphtheria complicating scarlatina. Nasal speech, appearing in the first to the fourth week, is often so marked after the healing of the ulcers, that speech grows unintelligible. Fluids, especially, are violently coughed out through the nose. More rarely total inability to swallow is found. As the disease is confined to the palate, the lips and tongue suffer no disturbance of function. But the soft palate hangs immovable, and shows no reflex when irritated. The soft palate does not participate in the enunciation of vowels, nor does it take part in the act of swallowing. A knowledge of this is sufficient to make the diagnosis.

GLOSSO-LABIO PHARYNGEAL PARALYSIS (laryngeal) must be distinguished from the foregoing. It was first brought to our knowledge by Duchenne's investigation in 1860, and is known also as bulbar paralysis, because the seat of the disease is in the bulbus medullæ. The first-mentioned name indicates sufficiently the extent of the palsy. As the palate is paralyzed, swallowing is as much interfered with as in the diphtheritic variety, but long before this stage is reached other symptoms gradually appear. The tongue shows marked impairment of function in chewing and speaking, so that the food can no longer be turned about in the mouth. Linguals can no longer be pronounced, consequently, some time before difficulty in swallowing is noticed, the patient's speech assumes a peculiar character, which may be imitated by pressing down upon the tongue with one finger, and attempting to speak with the finger still in the mouth. Frequent expectoration of saliva indicates the beginning of difficulty in swallowing. Inability to bring the lips into the position assumed in whistling, and a peculiar rigid physiognomy, are apparent. Paralysis of the glottis precedes death, which occurs in the first to the third year of the disease.

FOREIGN BODIES lodged in the esophagus form a chapter full of difficulties to the surgeon.

Therapy should be guided by diagnosis. But even if the diagnosis is assured, the therapeutic problems may still be insurmountable. It is readily seen how much greater they will be if, as often happens, the diagnosis is imperfect. In the cases collected by Professor Adelmann, four are mentioned in which no suspicion of a foreign body was entertained; in fifteen no positive diagnosis could be made, although all possible means were employed. In most instances we are obliged to rest content with the discovery of some obstruction in the esophagus which is proved to be a foreign body by the history elicited. In many cases the impact of the bougie will betray the nature of the obstruction, for it will show that no abnormal structure has been encountered. Foreign bodies, as a rule, are arrested in one of three spots: at the isthmus (level of third dorsal vertebræ), at the esophageal opening in the diaphragm, and at the cardia. Thin, pointed bodies frequently stick in the pharynx, and therefore this must always be carefully examined. Exploration of the esophagus proper is carried out by means of an ivory-tipped whalebone bougie. Duploy has suggested a flexible metallic bougie, provided with a silver olive at the anterior end and with metallic tambour at the other, which is to increase the sound produced by the impact against the foreign body. Esophagoscopy is a true blessing, especially in cases of impacted foreign bodies. Much information, and that of a positive nature, can be expected from it. Foreign bodies may not only obstruct the esophagus, but also *perforate* it. Perforation may be caused by the instruments used in searching for the foreign body. In addition, perforation may be due to the breaking down of a carcinoma, or to ulceration. How is perforation recognised? No general answer can be given to this query. Subcutaneous emphysema may signal the occurrence of rupture—another symptom being hemorrhage—not only bleeding from the mouth, but also the rapid formation of a false aneurism in the neck. Anadale found an aneurism of the inferior thyroid caused by a perforating foreign body. Perforation may also show itself by producing an acute mediastinitis or pleuritis.

A few cases of *spontaneous rupture* of the esophagus have been placed on record. As a rule, it occurred in strong men habitually addicted to alcohol, the accident taking place soon after a meal, and proving rapidly fatal. The esophagus was probably previously diseased. The symptoms were: retching and vomiting; then pain localized at the point of rupture (the cardia); premonition of impending death, collapse, grave respiratory disturbances, and subcutaneous emphysema, starting in the clavicular region and rapidly extending over the entire body.

CHAPTER XI

STENOSIS OF THE AIR-PASSAGES AND OTHER DISTURBANCES OF RESPIRATION

FABRICIUS AB AQUAPENDENTE, the successor of Fallopias, teacher of anatomy and surgery at Padua, said in praise of the operation of bronchotomy, that the physician who performed this operation was like the god Æsculapius. The operation is undertaken to furnish an artificial means of entry for the atmospheric air in cases of stenosis of the larynx or trachea which endanger life. The success of the operation naturally depends upon creating a passage *below* the point at which the stenosis is situated. This applies to all cases of laryngeal stenosis and to stenosis of the upper part of the trachea. Occasionally a tracheotomy opening is made *above* the stenosis, but only when dealing with a compression of the trachea, which may be relieved by passing an elastic or inflexible tube downward, in order to again distend the air-passage.

Tracheal stenosis may be divided into three groups:

1. Obstruction of the lumen from within—*Obturation.*

2. *Compression* from without.

3. Pathological changes in the walls of the tube which produce a marked narrowing of the lumen—*Stricture,* in the narrower sense of the word.

Obturation usually is produced by foreign bodies; but croup may be classed with this variety, because of the bulky false membrane produced by the process, which may cause a temporary but complete stenosis.

Compression stenosis may be due to foreign bodies in the neighbourhood of the trachea, extravasations, tumours arising from the thyroid gland, thymus, esophagus, lymphatic glands, sternum, clavicle, spinal column, and to aneurisms.

Changes in the wall of the trachea are manifold. The exudative processes of the mucous membrane, croup, and diphtheria, which narrow the lumen by producing false membranes, may be placed in this class also, for they are accompanied by swelling of the walls. Swelling in the parenchyma itself must also be included, as it narrows the lumen by the products thrown out (similar to abscess), or by the œdema which accompanies it; suppurative perichondritis, necrosis, and tubercular, syphilitic, and typhoid ulcers of the larynx (inflammations in the vicinity of the larynx). Chronic infiltration or thickening; cicatrization, following ulcerative destruction; and lastly tumours, which project into the lumen, belong to this class.

Cicatricial stenosis best typifies those rare cases where a slight stenosis, gradually progressive, becomes dangerous to life. As a rule, however, the danger occurs *suddenly*, so as to take both patient and physician by surprise. In the case of foreign bodies, a sudden stenosis is, of course, to be expected. In compression stenosis, urgent symptoms are usually caused by a catarrhal swelling of the mucous membrane of the larynx and trachea at the site of constriction. In dealing with inflammatory processes we usually expect stenosis, but the dangerous symptoms very often take the patient completely by surprise, corresponding to some unforeseen process in the inflammatory focus.

What are the characteristics of a dangerous STENOTIC ATTACK?

A typical attack of croup once seen is never forgotten. With the very first glance at a patient suffering from laryngeal stenosis we are impressed by the frightful efforts made by the little one. The child labours and strains; he fairly wrestles for air, and wildly endeavours to crawl up the wall, only to fall back among the pillows exhausted. Conscious of the obstruction, the little one tears at its neck and instinctively pries into its pharynx. Its face is a startling picture of anxiety and despair. Overwhelmed as it is, it utters neither cry nor scream, but lies absolutely speechless, giving vent only to deep, long-drawn, stridulous inspirations. Careful inspection discloses the fluttering of the alæ nasæ during deep inspiration, alternately widening and narrowing the nostrils. All the accessory respiratory muscles are driven to their utmost capacity; the neck muscles are on the stretch; the diaphragm constricts the lower thoracic zone, but is itself unable to descend. The epigastrium arches deeply inward with each inspiration. All, however, is in vain. Little air can pass the point of stenosis and reach the lung. The croup-membrane may hinder expiration also. If this is the case, the blood is forced out of the thorax at each expiration, and if this phase predominates the child grows cyanotic; otherwise, it is pale. After internal medicine has exhausted all its efforts, and the dyspnœa is still increasing, the surgeon is called in, and now has an opportunity to prove himself equal to Æsculapius. If tracheotomy affords no relief, the child grows quieter as the dyspnœa increases. Respiration becomes shallower, inspiration less audible, irritation of the periphery no longer meets with response, and carbon-dioxide poisoning causes death and puts an end to the agony.

The active exertions of the patient to obtain air; the full, noisy inspirations; the contraction of all the auxiliary muscles of respiration, and the deep pit in the epigastric region, are the most prominent symptoms of a stenotic attack of dyspnœa.

Surgery has its greatest triumphs in those cases in which the dyspnœa is caused by a FOREIGN BODY.

We are called in to see a child suffering with marked signs of stenosis. The history shows that the attack was severe from the outset, and that the child was in good health until the very moment at which the air-hunger first appeared. In such a case, the sole explanation possible is that a foreign body has entered the air-passages. Our first endeavour consists in introducing the hand into the patient's mouth in order to make sure that the foreign body has not lodged in the aditus ad laryngem, or, if it is there, to remove it. If it is not there, and time is too pressing to permit of laryngoscopic examination, tracheotomy must be performed as rapidly as possible. A child was brought to Schuh's clinic in great danger of asphyxia. Schuh was informed that the child had been in the best of health, but suddenly had had the attack after playing with pebbles. Without wasting a second, he grasped his knife and performed a tracheotomy. A small pebble at once popped out from the wound. If there is time to make an examination, the larynx should at once be inspected.

If this is impossible, auscultation of the larynx, trachea, and lung may in some cases be of service, for the foreign body may produce a characteristic sound as it flies up and down the passage and strikes against the walls. Blood-tinged sputum is of some value, as it points to injury of the mucous membrane resulting from the passage of a rough body. Subcutaneous emphysema is of even greater importance, as it indicates perforation of the mucous membrane. Louis, in the middle of the last century, already called attention to this symptom.

A similar attack may be caused by a foreign body arrested in the esophagus and pressing forcibly upon the trachea. It may happen, during a meal, that too large a bolus is swallowed and remains stuck fast. Not infrequently false teeth are swallowed and stick in the throat. Therefore, in all cases of sudden dyspnœa accompanied by marked cyanosis, it is advisable to explore the esophagus.

It is hardly conceivable that sudden, severe attacks of dyspnœa can occur in a previously healthy person unless they are due to a foreign body. Attacks due to neuroses of the larynx (*Spasmus glottidis*) occurring in young children, appear suddenly, but the attack may pass off, and the child again be lively and well before a physician can be summoned. In other instances, the dyspnœa may persist for hours. In most cases we are able *to judge from the external circumstances* whether we have to deal with a foreign body or not; as, for instance, in the case of a child that is attacked by the stenosis while in its mother's arms, without having played with any object. If short attacks have preceded, the disease is unmistakably characterized. Greater difficulty is encountered if the attack takes place during the course of an illness which has existed for several days. Here there is the possibility of laryngitis crouposa, catarrhal laryngitis, œdema of the glottis, or of a foreign body. Monti tells of a child that was supposed to be suffering with croup for three days. The child had a barking cough, croupy respiration, and was completely aphonic, and then developed a severe attack of suffocation. The examining finger, however, detected a small metal buckle which had become impacted in the glottis. But, as already emphasized, a foreign body can, as a rule, be taken for granted if a stenotic attack occurs without previous hoarseness and general malaise. Similar symptoms may, however, be noted in a child suffering from œdema of the glottis.

LARYNGITIS CROUPOSA and *laryngitis catarrhalis* are ushered in by fever, and stenosis is preceded by other warning symptoms. In some cases it is scarcely possible to distinguish one from the other; but, as the dyspnœa due to the catarrhal form never causes asphyxia, it is well worth while to know the diagnostic landmarks in order to be prepared for tracheotomy in the severer variety.

In *croup*, portions of the false membrane are either coughed up or can be seen upon the tonsils, and symptoms of a progressively advancing stenosis are usually present.

In *catarrhal laryngitis* the stenosis appears suddenly, but is of short duration. *Ascending croup* first gives the symptoms of an acute bronchitis, with the rapid development of cyanotic or cadaverous discoloration of the skin, and the evidences of carbon-dioxide poisoning. In the course of the following days laryngeal symptoms appear, just as in croup. Auscultation discloses inconstant râles, and small areas over which the respiratory murmur is inaudible, unaccompanied by any dulness.

Most of the children die, and but little relief can be obtained by performing tracheotomy.

An adult in the best of health may be attacked by the symptoms of stenosis. No swelling of the neck is visible; mouth, tongue, tonsils, etc., are normal. The patient is still able to talk. Sitting erect in bed, with neck stiffly extended, he briefly answers questions in a low, hoarse voice. Pain in the larynx, a change in the voice, or marked hoarseness and cough point to catarrhal laryngitis, the stenosis being due to collateral ŒDEMA OF THE GLOTTIS. The laryngoscope, which, as a rule, can be employed, shows the aryteno-epiglottidean folds distended into veritable sacs. If examination with the mirror is impossible, the finger introduced into the mouth can plainly distinguish these eminences when the tongue is strongly protruded. In such cases the patient's entire body must be examined, in order to find out whether œdema is present elsewhere, for people suffering with Bright's disease may have œdema of the glottis as the result of a catarrhal laryngitis. Such individuals show marked œdema of the lids after crying. Besides, it is well known that patients with kidney disease may have sudden attacks of dyspnœa, known as *Asthma urinosum*, and which is classified with bronchial asthma. Such cases are not infrequent. I saw a case in which, in addition to a moderate amount of compression stenosis due to goitre, severe attacks of dyspnœa occurred as the result of Bright's disease.

At the present day we use the term œdema *laryngis*, because the glottis is a cleft, and a cleft can not swell up. Boyle, who gave the first systematic description of the disease, believed the process to be a *non-inflammatory* serous infiltration of the submucous tissues of the glottis. Later, it was found that œdema of the glottis most frequently occurred as the result of inflammation, and in consequence of this advance in our knowledge the terminology was altered. Therefore we speak of laryngitis submucosa purulenta, or laryngitis œdematosa, or laryngitis phlegmonosa—terms which designate the underlying condition, and which are consequently more accurate.

Exceptionally, acute infiltration of the submucous tissues, occurring without any previous disturbance, may suddenly threaten the patient's life. This is most often due to a foreign body arrested at the rima glottidis.

Ziemssen records such cases. A labourer ate his meal in great haste, suddenly developed marked dyspnœa, and died. At the necroscopy a small spicula of bone was found wedged in the entrance of the larynx, producing œdema of the glottis. In another case the trouble was due to the rib of a tobacco-leaf; in a third, to a splinter of wood.

Frequently the œdema develops in a more subacute fashion, the stenosis increasing in the course of several days or even weeks. The development of the condition can be watched, but the course is very treacherous. In consequence of some small irritation, the gravest symptoms may take us by surprise.

A minute abscess in the vicinity of the rima glottidis may imperil the patient's life. The symptoms which precede the attack are, darting pain, increased on swallowing, pain on pressing upon the larynx, and a moderate irritation of the throat producing an irritative cough. Suddenly dyspnœa develops. Often grave fears are not entertained, and in many cases the danger is transitory; but at the next attack the dyspnœa may be so severe that only the most rapid interference will save life.

The significance of PERICHONDRITIS LARYNGEA varies in different cases. The anatomical situation of the process must be kept in mind. Türck, who has done exceptional work both in the pathology of the spinal cord and in laryngoscopy, has carefully studied the variations in the aspect of the disease which depend upon whether the focus is situated on the arytenoid, thyroid, or cricoid cartilages. These points would be very difficult for a beginner to determine, especially as the diagnosis is largely based upon the laryngoscopic pictures. The following points will prove of value: Perichondritis laryngea is, except in very rare instances, due to typhoid, syphilitic, tubercular, or carcinomatous ulceration. The diseases which are the primary trouble—namely, ULCERS OF THE LARYNX—must therefore be considered.

Typhoid ulcers are the least frequent variety encountered, especially at the present day, for the primary disease runs a milder course when treated by hydrotherapy. The process may be likened to the bedsores which appear over the sacrum and trochanter.

Tuberculosis of the larynx is usually secondary to a tubercular process of the lung; only in rare instances is it primary. The ulcers show great variety in their course; but if we regard their clinical manifestations in reference to stenosis, we may sum up the subject by stating that the more advanced stages of ulceration are met with on the parts most exposed to the irritations occasioned by deglutition and phonation. The ulcers are therefore situated on the mucous membrane covering the vocal cords, that of the cartilages of Santorini, or of the aryteno-epiglottidean folds. The cartilages are laid bare, the joints invaded, the parts exposed become necrotic, so that most of the upper portion of the larynx may be destroyed.

Syphilis manifests itself in many ways. It is seen as syphilitic catarrh, condylomata lata, gummata, and ulcerations. Stenosis is caused by the cicatrization of the deep ulcers which result from the breaking down of gummata. They cover large areas and may be situated upon the epiglottis, arytenoids, vocal cords, or tracheal cartilages. The resulting scars assume a great variety of shapes (ledges, projections, diaphragm-like membranes).

As a rule, no difficulty will be encountered in determining the nature of the ulcerative process which underlies a threatened or already present stenosis. Typhoid ulcers develop in the course of typhoid fever, while tubercular ulcers are present in individuals already suffering with tuberculosis of the lung. Sometimes the differential diagnosis between syphilis and tuberculosis is hard to make, especially if no foci are found in the lung. In such cases the following points are of value:

Syphilitic changes are usually painless; pain is felt only in rapidly spreading ulcerations upon pressure exerted from without, or upon deglutition. The painful period is always preceded by a noticeable change of voice, though both cough and swallowing remain entirely painless. As a whole, syphilitic destruction is characterized by its rapidity.

In *tubercular ulcerations*, on the other hand, a burning pain is felt from the very beginning of ulceration. The pain is greatly increased by speech, swallowing, and expectoration. The excruciating pain on swallowing is especially significant, for the patients, during eating and drinking, suffer the tortures of the damned.

Serious difficulty in swallowing occurs in the course of laryngeal syphilis only when extensive erosions of the epiglottis have taken place. In tubercular ulceration this difficulty appears early, and very frequently is the prominent symptom, so that the unbearable pain on swallowing is the patient's constant complaint.

NEOPLASMS OF THE LARYNX bear the most diverse relations to stenosis. In the most serious variety of new growth, *Carcinoma*, the symptom which usually precedes all others by a long period is hoarseness. If an individual past middle age suffers from an irregular and progressive hoarseness, which obstinately resists all treatment, the suspicion is immediately aroused that a slow change of texture is taking place in the vocal cords. Should tearing pains now occur spontaneously in the larynx, pharynx, and, what is well worth keeping in mind, *in the ear*, we are forcibly reminded of the lancinating pain of cancer. These pains are seen, for instance, in cancer of the thyroid gland, the patient suffering with radiating pain in the head and shoulder. The worst fears are justified if the symptoms of laryngeal stenosis gradually develop. At first, dyspnœa on severe exertion appears; later, slight efforts will bring on the shortness of breath which finally becomes constant. If a hard lymph gland can be felt in front of the sterno-mastoid, the diagnosis is almost assured, even without laryngoscopic examination. The decided emaciation of the patient, which is visible by this time, indicates also the malignant nature of the disease; and changes of contour in the laryngeal cartilages may be visible to inspection from without.

In dealing with a *Polyp* we reason differently. These tumours assume many and various shapes. They occur most often in very healthy subjects, but never in young people, always in adults. As the tumour is benign its growth is very slow. It is situated either on the upper or lower surface of the vocal cords, and acts as a continual irritant to the mucous membrane in the vicinity, so that this is in a continual state of chronic catarrh. Consequently a change of voice is invariably brought about, and the irritation is aggravated by exertion. At [illegible] can be observed. [illegible] the same symptoms [illegible] If we assume that [illegible] is always the possibil[illegible] change its position within the [illegible] Finally, it may fall [illegible] wedged fast. The [illegible] If, after the grad[illegible] of the above symptoms, sten[illegible] disappearing with equal rapidity [illegible] logical explanation, [illegible] body has been excluded, can be fur[illegible] growth. During one of these [illegible] the patient will frequently mention that he [illegible] there is a foreign body in his [illegible]

[illegible] from suffocation. [illegible] pick up an object from the [illegible] laryngeal route, by v. Bruns [illegible] vocal cord. It was pear-shaped [illegible]

[illegible] commonest new growths of the [illegible] associated with certain individual [illegible] in the afflicted subject. In the first [illegible] or at puberty (in contra[illegible] ; in the second, they usually [illegible] of anaemic and lymphatic habitus [illegible] polypi. The neoplasm may [illegible] surface of the larynx; the vocal [illegible] usual starting-point. The chief symp[illegible] are, disturbance of the voice, the [illegible] body, irritation causing cough [illegible] is relatively rapid in its progress

With the present facilities for making an examination, the diagnosis must be confirmed with the laryngoscopic mirror. But the practitioner will be able to make a provisional diagnosis, without further examination, by means of the symptoms outlined above.

Paralyses offer an interesting field, which is, however, open to the specialist alone. Especially important is *paralysis* of both *posterior crico-arytenoid muscles,* which prevent the glottis from opening during inspiration, thus producing danger of suffocation. The disease develops insidiously, usually unaccompanied by catarrh or alterations in the voice. Noticeable is the fact that expiration is not disturbed—therefore no mechanical obstacle is present.

In contrast to this, *bilateral paralysis* of the *recurrent laryngeal nerve* can be diagnosed almost positively without laryngoscopic examination. This disease is marked by complete aphonia, absence of dyspnœa during quiet respiration, inability to cough or expectorate vigorously, the patient, however, being able to expel a powerful blast of air from his mouth.

Hysteria is a true disease, yet hysterical women, "perfectly well" in the afternoon, may in the evening be attacked by dyspnœa, which simulates stenosis of the air-passages. These attacks are rarely serious; as a rule, the true state of affairs is readily unmasked. The hysterical facies, the rolling of the eyes, the simultaneous appearance of dysphagia (this is the rule in hysterical subjects), the rumbling of the intestines, the occasional distortion of the face, the cramps, etc., all give the picture of an hysterical attack. If we succeed in eliciting some answer from the patient—often a task demanding experience and *savoir-faire*—the voice at once shows the absence of laryngeal trouble. At times the patient, craving sympathy, voluntarily asks, in a low voice, whether she will die of suffocation. Although not dyspnœic at this moment, a fresh attack may suddenly make its appearance without warning.

I often cure such an attack (after having taken the relatives into my confidence) by prominently displaying a few instruments upon the table, and declaring that an operation will be necessary if the disease grows worse. The attacks then fail to recur.

A stenotic attack can occur in an apparently healthy individual, independent of any mechanical obstruction of the air-passages. I was forced, on one occasion, to perform a tracheotomy on a young girl who was in great danger of death from suffocation, although I was unable to find any œdema of the glottis. She was relieved, but pneumonia developed on the following day, and the patient died. Larynx and trachea were found free, and no trace of œdema could be discovered, but there was *marked hydrocephalus*. Such cases are at present obscure. I saw an analogous case in an insane female at Innsbruck.

The diagnosis of *tracheal stenosis* due to pressure exerted by tumours, especially by goitre, is more difficult. One class of cases is represented by women afflicted with goitre, who suffer from dyspnœa during the menstrual period, although they are troubled with only the slightest discomfort at other times. Such cases are unassociated with any danger; cold compresses about the neck reduce the hyperæmia of the gland and palliate the discomfort. In another class of cases a very slight degree of stenosis of the trachea may cause asphyxia, but the explanation must be sought elsewhere than in the trachea itself. The younger Demme has made valuable observations and studies which demonstrate the fact that neuroses of the larynx may occur in goitre through the agency of the recurrent laryngeal nerve, simultaneously with tracheal compression. This was known to be the case in carcinoma, as can be seen by consulting Lebert's references on this subject. Very lately, Professor Rose has emphasized the danger which may result from the pressure exerted by goitre upon the trachea, so that we may correctly speak of death from goitre (*Kropftod*). The cartilages of the trachea suffer a change of texture (a softening) which causes the windpipe to lose its circular shape and become a mere slit, usually likened to the scabbard of a sword. The ordinary movements of the neck suffice to distort the softened trachea so as to reduce its lumen to a minimum.

We must remember that the picture can be complicated by various other factors. In addition to the compression of the trachea, an agglomeration of lymph glands may press upon one of the bronchi, or respiratory disturbances may be secondary to disease of the lung. The symptoms can be differentiated only by a careful physical examination of the thorax. If stenosis occurs in a goitrous subject, our first care must be to ascertain whether the condition is due to compression. The following factors should be borne in mind: 1. Careful examination of the mouth and pharynx excludes any disease which might produce stenosis of the larynx. Testing the voice excludes laryngeal affections. In pure stenosis aphonia is wanting, and the changes in the voice which are present differ from the changes seen in primary laryngeal affections. If a laryngoscopic examination is feasible, it will at once decide whether or not the larynx is narrowed; in favourable cases a skilled laryngologist may actually *see* the stenosis of the trachea. 2. The position of the larynx and trachea should be examined. If great displacement or distortion is found, auscultation at this site should be practised. The whistling sounds heard during respiration are characteristic. 3. One must not forget that the tumour, although of small size in the neck, may extend over a large area behind the sternum. This must be established by percussion over the sternum. 4. The history will show whether *dysphagia* existed for any length of time, in which case a retrotracheal extension of the tumour may be taken for granted. More rarely, passing of the esophageal bougie confirms the diagnosis. 5. Catheterization of the trachea will clear any remaining doubts if the laryngoscopic examination is negative.

Auscultation requires special emphasis. The whistling sound is loudest during inspiration, differing somewhat in this respect from the sounds heard in catarrh, asthma, and emphysema, which are most marked during expiration. The quality of the sounds are very similar. In addition, the whistling inspiration is heard most distinctly in the median line anteriorly, and between the scapulæ posteriorly.

The presence or absence of complications must next be determined. This question is of paramount importance in high degrees of stenosis, in which the advisability of performing tracheotomy is under discussion, for if the lumen beyond the trachea is narrowed, a tracheotomy would be of no avail. Such a condition may be assumed to exist if one lung does not breathe, because the bronchus is occluded or compressed. The same holds true if, in addition to substernal dulness, there is diminished breathing and a lessened circumference of the thorax on one side, accompanied by whistling sounds at the site of the supposed stricture.

All in all, it is well to remember that two points alone are of real importance. In the first place, it is necessary to recognise that a stenosis is present. The loud stenotic sound occurs in this condition alone, and thus characterizes the breathing of stenosis. In all other respiratory disturbances, whether due to reduction of the lung surface (œdema, pneumonia, pneumothorax), or to lessened respiratory movement (emphysema, spasm, or paralysis of the diaphragm, widespread paralysis of the muscles of respiration), or to circulatory disturbance, the stenotic sounds are absent, although orthopnœa is not wanting. The stenotic attack is marked by *stridor*, the asthmatic by *stertor*. In the next place, the diagnosis of the underlying disease, as a rule, is the deciding factor. It determines whether or not surgical interference is to be of use; if the stenosis is not within the scope of the knife, it becomes a medical case.

CHAPTER XII

INJURIES OF THE THORAX

DISCUSSION of injuries of the thorax had better be considered from the point of view of actual practice. If called to a patient suffering from an open wound of the thorax, the treatment differs radically from that used in a subcutaneous injury. In the former case we must be prepared for immediate interference, in the latter we may safely wait, for these injuries usually run a much more favourable course, unless immediately fatal.

Especially important is the fact that the lung, heart, great vessels of the thorax, and the arteries of the thoracic wall (internal mammary or an intercostal) may be torn even in *subcutaneous* injuries. Not only these organs, but also the diaphragm, liver, and spleen may suffer considerable damage without any external wound. It is readily understood that such injuries are usually caused by the dislocated fragments of fractured ribs. If the chest is crushed between the buffers of two cars, or the patient run over by a heavily loaded truck, or caught underneath a barrel during unloading, or if in a street fight one of the combatants falls and is kicked and stepped upon, it is evident that these enormous and sudden forces drive the costal fragments deep into the thoracic and abdominal cavities. Thus are caused bruises, tears, rupture, and perforation of the most important organs. Although rupture of the lung and of the heart not due to an impacting fragment have been observed, such cases are exceptional. Rupture of the organs must then be ascribed to the compression of the thorax. Our observations will be confined to cases due to fracture of the ribs and sternum.

The first question naturally follows: How is a FRACTURE OF THE RIBS recognised? Most easily recognised is a comminuted fracture, in which one or more fragments are broken off from the rib; for in this variety the most important sign of fracture—false point of motion—is visible on inspection. During expiration and inspiration the two sharp, serrated ends of the ribs, respectively anterior and posterior to the site of fracture, raise the skin and threaten to burst through. The intermediate fragment lies in a hollow, and makes much smaller excursions. In such cases, by carefully listening, short, grating, crepitant sounds are audible. The hand can press the fragment, which has been broken out, inward, and also move it upward and downward. If several ribs have been fractured in this fashion, the condition can be recognised from a distance. The thoracic wall at the site of injury is flattened, and the fragments can be felt at this spot.

Simple fractures of the ribs, in which there is a prominence or a depression at the site of fracture, are as readily recognised as the comminuted variety. In cases of simple fracture, a spinelike projection along the line of fracture juts out from the convexity of the thorax, or, if the dislocation is inward, a perceptible hollow results.

No greater difficulty is encountered in the recognition of the condition if the ends of the fragments lie [illegible] of ribs [illegible] of service: 1. The scap- [illegible] the seventh ribs; the breast [illegible] the sixth. 2. The tip [illegible] of the ninth rib if [illegible] the side. 3. The [illegible] rib. With the aid [illegible] of ribs can be deter-

A case in which a physician diagnosed the fracture of a rib was brought into court. He based his diagnosis upon the fact that the patient had felt pain when the anterior and posterior ends of the rib were pressed upon *simultaneously*. I impugned the correctness of his conclusions, because pain would be caused by pressure at the site of injury even in a contusion. The physician should have pressed upon the posterior end alone in order to see if pain was caused anteriorly.

FRACTURES OF THE STERNUM require but little mention. Longitudinal fractures are extremely rare. Transverse fractures and diastases between the manubrium and gladiolus, and between the gladiolus and ensiform can not be recognised if displacement, abnormal mobility, and crepitus are wanting, or if a swelling of some size obscures the line of fracture. The swelling may be due to emphysema, blood, or, at a later stage, to abscess. Fracture has probably occurred if the patient heard a cracking sound at the time of injury, if he feels no discomfort with the head flexed and the body inclined forward in a semi-erect position, but suffers violent pain at a fixed point on raising the head, coughing, or pressure upon one end of the sternum. If malformations—i. e., changes of contour of the sternum—are found, unaccompanied by signs of mobility, they do not in themselves speak in favour of fracture, because other causes may have been at work—e. g., congenital longitudinal fissures. In the cases in which displacement, crepitus, mobility, or at least increased resiliency of one piece is present, the general rules apply, and further discussion becomes superfluous. The objective signs are unmistakable. One thing alone must be remembered: that is, to pay attention to the position of the ribs. If, for instance, one or both of the second ribs are dislocated from their sternal attachment, and signs of a transverse fracture exist, a diastasis has taken place between the manubrium and gladiolus. In cases of diastasis between the body of the sternum and the xyphoid appendix (which, by the way, frequently is the seat of distortions or anomalies), it has been noticed that the patients vomited when the appendix was pressed inward toward the abdominal cavity.

Subcutaneous injuries to the *heart* and *great vessels* are, as a rule, instantly or rapidly fatal. " A woman was caught between a wall and a heavily loaded truck, and died almost instantaneously. Several ribs were found broken and driven into the lung; the pericardium was distended with blood; the superior vena cava almost completely torn across, and separated from the auricle." This is one of many examples found in the literature. In isolated cases death does not take place until several hours have elapsed.

Theoretically, the following could stand as typical of a ruptured *intercostal artery:* A progressively increasing area of dulness following the injury; a buzzing sound at the site of rupture, and the symptoms of increasing internal hemorrhage. This picture ought to suffice for the diagnosis, but it is purely imaginary. The diagnosis of the rupture of an intercostal artery can be made at a later date if an aneurism, which can be pushed back into the chest, develops. Such a case has occurred.

The recorded cases in which the *diaphragm* was ruptured do not permit us to formulate any rules for their diagnosis. One case, in which the *liver* was injured by a fractured rib, could be diagnosed with certainty, because the fracture was compound and the liver exposed. In another instance the liver was injured in a subcutaneous fracture of the rib. Although the liver suppurated, icterus and chills failed to appear.

Subcutaneous INJURIES OF THE LUNG AND PLEURA are among the commonest accidents which result from the severer forms of violence to the thorax. They admit of fixed rules in diagnosis. If blood is coughed up during the course of several days, in consequence of such an accident, and if no further symptoms arise, although a rib has been broken, we may assume that the lung parenchyma has been torn or bruised. This may take place without rupture of the visceral pleura; but we can not be positive that the pleura has escaped, because a pneumothorax of slight degree may disappear very rapidly. If *pneumothorax* or hæmo-pneumothorax can be demonstrated, it is evident that the pulmonary pleura has been injured. Has the costal pleura been torn? If a fracture of the ribs has taken place, the costal pleura has been torn. One case, however, is on record, in which the third and fourth ribs on the left side and the third rib on the right were fractured on their outer surfaces only; the costal pleura was intact, and yet pneumothorax of the right side developed, owing to rupture of the lung. In practice we may assume that the costal pleura has been injured by the ribs in cases of pneumothorax. Assurance is made doubly sure if subcutaneous *emphysema* appears. The behaviour of the emphysema varies. If it appears and spreads rapidly, the pulmonary and costal pleura are both usually injured, but commonly no air will be found in the pleural sac. For in those cases in which the pleural cavity is shut off by adhesions at the site of rupture, the emphysema is wont to develop more rapidly; often, in the course of half an hour, extending over the entire body. But no hard-and-fast rule can be laid down. Rapid spread of the emphysema may take place with pneumothorax, which, however, can not be discovered if the emphysematous swelling covers the chest. Sometimes the emphysema appears as a circumscribed swelling which is inflated at every inspiration, and alternately rises and falls, while the emphysema at the same time continues to spread along the neck, head, arms, scrotum, etc. Injury to the lung and pleura, therefore, causes a great variety of symptoms—hæmoptysis, hæmothorax, pneumothorax, and emphysema variously combined.

A few minor points may be added. It is well known that fracture of a single costal cartilage is a rarity. Rarer even than this is the dislocation of a rib at the sternocostal junction, and also the dislocation of the two lower true and three upper false ribs from their respective costal attachments. There may be uncertainty whether the break is situated at the junction of cartilage and rib—so-called diastasis—or at the end of the bone close to the cartilage, or in the cartilage close to the bone. This point may safely be left in doubt, and attempts to clear the question for the sake of diagnosis, by means of akidopeirastik (investigation with a needle), are unjustifiable. The callus situated near the end of the cartilage, resulting from a fracture, ought not to be confused with a rachitic enlargement of the ribs—the so-called rosary—even if no history can be elicited; for the rosary is bilateral, and is accompanied by flattening of the thorax, in addition to signs of rickets elsewhere in the body.

Fracture of the ribs has been caused by muscular action. Severe sneezing, due to taking snuff, occasioned it in one instance. Fracture of the sternum due to muscular action occurs, as, for instance, during the pains of labour. This fact is generally known, as it is mentioned in all anatomical lectures. If the force was noticeably weak, it is well to remember that fragilitas ossium may be present. In old people, it is advisable to give a guarded prognosis if the case is not seen before the callus has begun to form, because a carcinoma of the ribs might cause the fracture, and then continue to increase.

Large extravasations may be found on the wall of the thorax as the result of some injury to the shoulder. Pitha reports a very interesting case of this nature. An extravasation the "size of a loaf of bread" was situated on the thoracic wall after an injury to the shoulder. Autopsy showed, omitting other details, that the subscapular artery had been torn away from the axillary trunk.

We now turn to the discussion of OPEN WOUNDS OF THE THORAX.

The wound is unmistakably penetrating:

1. If part of the lung protrudes (prolapsus pulmonis). In recent cases such lung tissue is readily recognised because it contains air, and therefore crepitates; later, the lung is incarcerated and becomes gangrenous. If in the projecting tissue a sharp margin or incisure can still be recognised, it will aid in making the diagnosis.
2. If air streams in and out of the wound.
3. If pneumothorax or hæmo-pneumothorax exist.
4. If a penetrating body, which by its position and length shows that the wall of the thorax must have been perforated, is removed from the wound.

The case may be doubtful if emphysema, *and that alone*, is seen; for, as has been shown, emphysema may occur in non-penetrating wounds as a result of the movements of the chest wall aspirating air into the wound. In such cases the wound should be promptly closed, and probing avoided.

As a rule, emphysema indicates a penetrating wound, as aspiration of air into the wound is a rare complication; if present, the resulting emphysema is very limited in extent.

The case may also be doubtful if no pneumothorax exists, only coughing of blood. The pneumothorax may have disappeared. A penetrating wound, in such cases, is only probable; and the probabilities must be carefully weighed and all circumstances considered before an opinion can be formed.

If the lung is adherent at the point of injury, hæmoptysis may occur, and yet pneumothorax be absent. As a rule, however, emphysema will then be found.

If a wound proves to be penetrating and there is considerable hemorrhage, we may suspect that an intercostal artery or the internal mammary, if the wound is at the sternum, has been damaged. Suspicion is strengthened if the hæmothorax increases and symptoms of internal hemorrhage develop; but the diagnosis must remain in doubt unless we see the spurting vessel in the wound or feel the warm stream of blood when the finger is introduced at the proper site.

George Fisher has collected and critically compiled all the cases of *wounds of the heart and pericardium* which appear in the literature. His work shows that there is not a single pathognomonic symptom which permits the diagnosis of a wound of the heart with any certainty. Dupuytren describes a case where a patient came to the hospital with five inches of steel sticking in his heart. He evinced neither pain nor dyspnœa; his pulse was quiet, his walk firm, and his facial expression unchanged. The patient died after twenty-two hours. The right auricle and the lung were wounded.

The pericardium or the heart are injured beyond a doubt if the wounds of these organs can be felt or seen. Bamberger, in a wound of the pericardium, was able to palpate with his finger the shape and change of position of the heart due to its contractions. Ollenroth was able to do the same, and in addition saw a flat wound about six lines in length, at the apex. The diagnosis of complete avulsion of the heart—it has happened that the detached heart has popped out of the wound—is an anatomical and no longer a surgical task. Formerly, surgeons claimed that probing of the wound, to determine whether the pericardium or heart had been injured, was contraindicated. The location of the wound, the primary symptoms—such as extreme terror, unconsciousness, and the forebodings of impending death—and the later ones—such as fainting, subnormal temperature, trembling, cold sweat, combined with the information obtained by auscultation, percussion, and observation of the pulse—sufficed in many instances to make a probable diagnosis. In more modern times wounds of the heart and pericardium have been actively treated. The wounded pericardium or heart has been successfully sutured. In order to employ such therapeutic measures, diagnosis of the conditions must necessarily follow certain rules. It is no longer contraindicated to enlarge the wound sufficiently to introduce the finger, or, in a given case, to expose the heart by resection of the ribs, in order to ascertain the true state of affairs.

Wounding of the *pericardium* can be positively diagnosed without the use of probe or finger, if pneumopericardium is found and a wound of the pleura can be excluded. The air enters with the instrument which causes the injury. By very exact deductions, wounds of the heart can be distinguished from wounds of the great vessels.

Rupprecht reports the case of a painter, who stabbed himself up to the hilt with a dagger, in the region of the heart. The dagger was withdrawn and dark blood flowed from the wound. As the blood was not frothy and no air came out of the wound, injury to the lung was excluded. None of the venous trunks situated in this neighbourhood could have been injured without the lung suffering at the same time. Therefore the diagnosis of injury to the right ventricle was made.

Injury to the *esophagus*—as the result of bayonet or knife thrusts—are positively recognised as such when the ingested food flows from the chest wound. If this sign is wanting, such an injury may be suspected with considerable certainty if an area of dulness, which at first might be due to blood or exudate, regularly and markedly increases after the patient eats or drinks. The same holds true if the patient feels a peculiar sensation on swallowing, accompanied at first by *vomiting* of blood, and later of pus.

Wounds of the *diaphragm* can be suspected from the pain, caused by the stronger contractions of that muscle, due to coughing or vomiting, and from the severe hiccough which follows; but it is evident that the appearance of these phenomena in a penetrating wound of the thorax can admit of other interpretation. A large tear in the diaphragm can alone account for the presence of abdominal viscera in the thorax—in other words, a diaphragmatic hernia. But reflection will show that the proof is very difficult. If any of the hollow viscera enter the thorax, the resulting tympanitic note and displacement of the heart admits of more than one explanation, as pneumothorax already exists. If the stomach is forced into the thorax and displaces the liver, the consequent displacement of liver dulness toward the region of the stomach would favour the diagnosis. Symptoms of incarceration, which will be spoken of under hernia, would lead to careful analysis and weighing of the facts.

The diaphragm may be penetrated in thoracic injuries, the wound extending directly into the abdominal cavity. In such cases omentum may prolapse through the diaphragmatic wound into the thorax, and appear externally through the thoracic wound. The presence of a combined thoracic and abdominal wound is then apparent at a glance.

The primary symptoms which follow *gunshot wounds* of the thorax may be exceedingly mild, even if the projectile has passed through the lung. The course of the bullet can not be accurately determined, and therefore prevents an early diagnosis. As pneumothorax and emphysema fail to appear, hæmoptysis is the guiding symptom. In many instances insignificant primary symptoms are followed by widespread and frightful gangrene if septic material has been introduced by the projectile. The situation of the bullet can not always be determined. As an instance of a lucky diagnosis, I will mention a case of Strohmeyer's. Here, in addition to a pleuritic effusion, pain was felt in the neighbourhood of the spinal column, and spasm of the diaphragm followed. He declared that the bullet was at the floor of the thorax, and autopsy confirmed his view.

CHAPTER XIII

TUMOURS OF THE THORAX AND BREAST

Abscesses of the Thorax, whether acute or chronic, may be of greatest interest to the diagnostician. The following remarks are of special importance to the beginner, and well worth careful study: In the first place, the question whether an abscess or some other cystic swelling has to be dealt with will arise. This question would be of special importance if the diagnosis lies between an abscess and an aneurism. Such a case is possible, as an abscess in close proximity to the heart may pulsate, and, on the other hand, an aneurism of the thorax may neither pulsate nor produce a bruit. If the tumour pulsates, we must ascertain whether it is compressible, or whether it is not merely raised up during the arterial diastole instead of being increased in volume. If no decision can be reached, and the history gives no aid, an exploratory puncture with the finest needle, although not entirely free from risk, should be undertaken with the precaution of drawing the skin to one side, so as to avoid bleeding when the needle is withdrawn.

Other conditions requiring differentiation are chronic abscesses and mucous bursæ. I saw one case in which fluid had collected beneath the pectoralis major. A chronic abscess always is preceded by some inflam matory symptoms, and some part of the bony framework is sensitive, either a rib, the clavicle, or scapula, etc. The cold abscess, further, is painless, and occurs in individuals of lymphatic temperament. An abnormal bursa is more sharply circumscribed and flatter; the period of development is slow, extending over years; the spot is exposed to friction; the bones are painless; the patient is strong and muscular.

The following case is suggestive: A swelling which gives a *tympanitic* percussion note appears after trauma. It may be a hernia of the lung, which is characterized by being reducible. It may, however, be an abscess containing air. In this given case the swelling can not be reduced, but percussion and palpation both give a splashing sensation, which indicates that the contents is composed of both air and fluid. Such abscesses result from the perforation outward of a cavity. At times this condition is seen in psoas abscesses. Before the abscess points at the groin a second pocket may show in the lumbar region. This part of the abscess may lie over the lowest ribs and contain gas, as is sometimes seen in a subserous abscess. The gas reaches the abscess from the intestine by diffusion; or the abscess may have perforated at some distant point and aspirated the air. But all these are very rare conditions.

If the various symptoms favour the diagnosis of abscess, the next question to be solved is, From what layer does the pus take its origin? The abscess may have started in the pleural cavity—a perforating empyema; or in the subpleural space, a peripleuritic abscess; or from a rib or from the sternum, as in osteomyelitis and tuberculosis of these bones. It may also begin in the connective tissue between or behind the muscles, or in the subcutaneous connective-tissue layer.

To decide the depth of an abscess the following points will be found of service:

An abscess superficial to the layer of muscle is very prominent over its entire surface. The muscle can be felt only by deep palpation, and appears to lie beneath the fluid. Active contraction of the muscle renders the abscess more prominent.

An abscess placed below the layer of muscle is less prominent; skilled palpation detects the muscle bundles, which pass over its surface. The fluid is recognised only by deep pressure. If the muscle is contracted, the abscess is less evident, because the muscle squeezes it flat. At the same time the tension within the abscess is considerably increased.

A striking demonstration of the submuscular site of an abscess may be obtained if the abscess has made its way into the subcutaneous tissue at one or two spots. If the muscle is relaxed, that part of the abscess which has broken through can be pressed back beneath the muscle. If the muscle is now contracted, the reduced portion reappears, and is found very tense.

Abscesses attached to bones naturally possess all the signs belonging to submuscular abscesses, but in addition are firmly *attached*, and in some cases encircled, by a ridge of bone. As abscesses of the connective tissue, however, are rarely situated beneath the muscles, it is safe to say that most submuscular abscesses start from bone; this applies especially to the so-called gravitation abscess. An abscess may be found beneath the pectoralis major, but examination fails to detect a ridge of bone which might show the connection between the abscess and the underlying bony parts. We then attempt to determine whether or not the abscess is movable on the deeper parts. An immovable abscess usually originates from the bone. But not infrequently the abscess has burrowed down so far from its starting-point (a cavity in a rib or vertebra) that it is now connected with the original focus by a mere fistulous tract. If the pus finally reaches the superficial layers, the abscess may be movable, and yet be derived from bone. Occasionally such cases do occur, but more often the abscess travels along the deeper layers and is immovable, even if its communication with the bone cavity is narrow and long. In most cases origin from some bone can be claimed for the abscesses, but the bone is usually far removed. Therefore the ribs above the abscess, the clavicle, the coracoid process, etc., should be examined for swelling and tenderness. The history will show that pain first appeared at this distant spot. If an abscess starting from the bone has destroyed the muscle which covered it, the usual method of determining the depth of the abscess is no longer applicable. The palpating finger fails to meet with the resistance offered by the muscles, but sinks in deeper, and may detect a bony ridge if present. This sign is of special value in tubercular abscesses, which frequently lack the bony ridge. Collections of pus beneath the clavicle demand careful examination of the sternoclavicular joint for abnormal mobility and crepitus, because the diagnosis is more complete if the destruction of this joint has been demonstrated.

Abscesses which point from within the chest—i. e., empyema and peripleuritic abscess—come under observation when they grow prominent externally. A *peri-*

pleuritic abscess is distinguished from an empyema by the following signs: only those intercostal spaces are widened which are in the immediate neighbourhood of the fluctuating tumour. The more distant spaces are, if anything, narrowed by the crowding together of the ribs. The tension of the fluid increases during expiration. The dull area has not the line of demarcation so characteristic of pleuritic effusions. These symptoms are made all the more prominent if auscultation and percussion below and above the dull area prove that the lung properly performs its function. Finally, the peripleuritic abscess does not cause displacement of the organs.

The diagnosis of an abscess of the thorax wall is rendered more difficult if situated behind the female mamma. To differentiate a neoplasm of the mamma from an abscess of the ribs becomes doubly difficult under these conditions. The mobility or fixed position of the swelling will be decisive, but this very sign is frequently wrongly interpreted. The tumour may appear immovable owing to faulty examination, and the diagnosis of a neoplasm is made. In order to recognise immobility, carefully palpate the whole circumference of the tumour with the finger-tips. If the mass can not be crowded away from its attachment, and remains immovable, try to establish the independence of the gland and the tumour by keeping the one hand on the base of the swelling, at the same time attempting to move the breast with the other. The only query still to be answered is whether the growth is a neoplasm springing from the ribs or an abscess of similar origin. This will be decided by fluctuation, sensitiveness of the ribs, the history, etc.

An *Actinomycotic focus* derived from the fungus which has entered the lung, produced an adhesive pleurisy, and then penetrated the thorax wall, may offer considerable difficulty in its diagnosis. Its course may be exactly similar to that of a small cold abscess springing from a rib. If the individual is robust, this contradiction will arouse our attention. If the abscess is large, it will show—corresponding to actinomycotic phlegmon in the neck—the signs of hard infiltration about its periphery combined with central fluctuation and noticeable absence of all pain.

ABSCESSES OF THE MAMMA are instructive to the beginner. Velpeau, who had an excellent and experienced eye for detail, distinguished three forms, which we will now consider:

Subcutaneous abscess, situated in front of the gland, projects from the breast like a smaller hemisphere placed upon a larger. It develops rapidly, soon grows soft over its whole surface, and points within a short time. *The breast does not grow hard and tense.*

An *abscess in the substance of the gland* is first noticed as a hard, diffuse nodule within the breast. The skin gradually becomes adherent at some point, and grows redder and redder. The pain is intense, the redness of the skin increases in extent, and fluctuation is more and more readily demonstrated. The whole breast participates in the change of outline, and finally the apex of the swelling corresponds to the point of maximum fluctuation.

A *retromammary abscess* may be suspected by the changes noticed on inspection. The position and shape of the breast are changed. The gland is erect, and does not hang down like that of the opposite side; its form is more hemispherical. Fluctuation should be sought for *above* the gland. In more subacute cases the following striking demonstration, which aims to show the change in fluctuation, can be attempted. The lower part of the breast is squeezed against the chest-wall, and fluctuation is more apparent at the *upper* margin of the gland. If the upper quadrant is crowded against the thorax, fluctuation is felt at the *lower* margin. Such abscesses are opened above the gland, and not, as usual, at the most dependent portion; for, if the opening is made below, the gland sinks against the chest-wall and the pus can not drain. This may be obviated by introducing a stiff drainage-tube, but opening the abscess above is the more rational treatment. If the abscess is very acute, it may burrow in the cellular tissue as far as the sternum.

TUMOURS OF THE BREAST are of frequent occurrence. They are superficially situated and readily demonstrated, therefore usually chosen in clinical instruction to school the beginner in the diagnosis of tumours.

Indications of *malignancy* are evinced by the fact that malignant tumours infiltrate neighbouring tissues, regardless of their structure, and slowly replace them. A malignant growth of the breast advances toward the skin, backward toward the pectorals, the deeper layers of the thorax wall, the ribs, finally encroaching upon the pleura and lungs. This unlimited growth takes place with great relative rapidity. A cancerous node will reach a certain size—let us say that of an egg—in the course of months; a lipoma would require years to attain similar proportions. Malignant neoplasms, at an early date, infect the neighbouring lymphatic glands

—that is, the glands to which the lymph-currents of the diseased breast flow.

A well-developed cancer of the breast will give the following picture: The tumour is the size of a goose-egg, hard in consistence; parts of its periphery are sharply circumscribed, and other parts merge into the gland tissue. The mass is placed in the outer quadrant of the breast. Part of the tumour, covering about the area of a walnut, has ulcerated. The ulcer shows an excavated base covered with a dirty exudate, and indurated, bright-red, irregularly granulating edges. The skin about the ulcerating area is somewhat thinned, and can no longer be raised into folds. The nipple is broadened and retracted. The tumour is movable in all directions. In the axilla a group of hard, round, movable, and entirely painless glands are felt. The disease has existed one year, the ulceration only a few weeks. In this case malignancy is evident, because the skin has been attacked and has ulcerated, the axillary glands have been infected, and the rate of growth has been rapid.

The above description does not indicate whether the tumour has invaded the structures behind the breast. The ribs have certainly not been reached, for the tumour is movable, but the pectoral muscles may have already been infiltrated and incorporated. This can be shown in the following way: If the pectorals are relaxed, they move with the tumour, and this will consequently seem movable; but if the muscles are passively stretched, the tumour will be less movable. This is especially evident when we attempt to move the growth in the direction of the muscle fibres; much less so if movement is made transversely to the fibres, for the muscle can never be made tense to such a degree as to prevent all motion in a direction transverse to its long axis.

Let us take for granted that the tumour has infiltrated the pectoral muscle: thus its extension in a second direction has been proved. If the tumour had been examined three months previously, it would have most likely been movable, even with the pectoralis passively stretched, by maximal abduction of the arm. But, at this early date, malignancy would have been shown by the immobility and the thinning of small areas of the skin. At a still earlier period, symptoms of malignancy, such as attachment of the skin and enlarged axillary glands, would probably have been wanting, but retraction of the nipple might have been noted. This last symptom is due to the fact that the tumour involves the ducts of the gland at an early stage. A scirrhus atrophies and contracts in its centre, and thus pulls upon the ducts like a driver upon the reins, with the result that the nipple becomes less prominent.

The contraction which takes place in scirrhous carcinoma changes the position and appearance of the whole gland. The opposite breast hangs flaccid, while the affected one is usually pulled upward. Not infrequently the surface of the gland shows a small depression—the so-called cancerous umbilication—at a point at which the skin has become attached and retracted. Instead of this, the skin may be wrinkled at one or more points, as the result of the attachment of several parts of the skin, with subsequent cicatricial contractions. The position of the organ, the retracted nipple, the depression and wrinkling of the surface (known in English as "pigskin"), all show the central atrophy of the growth.

Carcinoma of the breast appears in women at about the age of forty years; less commonly in the third decade. In younger individuals carcinoma need not be considered in the differential diagnosis.

At times a chronic *mastitis*, the result of some previous puerperium, may cause considerable difficulty in its differentiation. I have seen surgeons whose experience covered thousands of breast tumours hesitate. In a given case, if the woman has reached the age at which carcinoma is common, if the tumour is not sharply circumscribed, if its resistance is considerable, and no hard, painless glands are present, the uncertainty in diagnosis is great. Suspending the breast with an appropriate bandage, application of a moderate amount of cold and an iodide salve, will do no harm to a possible cancer, and will be of benefit to a mastitis. The behaviour of the nipple is of great importance in these cases. A marked retraction speaks strongly in favour of cancer.

Sarcoma of the breast is of much rarer occurrence than cancer. It shares its rapid rate of growth with cancer, but, unlike this neoplasm, it ulcerates much later. A cancer which is half the size of the breast is, as a rule, ulcerating; a sarcoma which has reached the size of a child's head may still be subcutaneous.

Cystadeno-sarcomata represent the giant tumours of the breast; they may attain enormous proportions. The presence of markedly fluctuating protuberances, mobility upon the deeper parts, and slow rate of growth are observed. In such giant tumours single cysts may rupture; the skin covering others may be injected and very tense: isolated axillary glands infected, but the tumour remains movable on the muscles. Smaller cystosarcomata are diagnosed by the lobulated surface, with scattered areas of fluctuation, mobility, and sharply circumscribed periphery. At a very early stage the sharp circumscription and the lobulated surface serve to distinguish this variety.

In younger women, small, sharply circumscribed, elastic, and extremely movable *adenomata*, often multiple, may be found either at the margin of or in the substance of the mammary gland.

TUBERCULOSIS of the breast is not as rare as was formerly supposed. A superficial, tubercular ulceration of the skin may be seen quite often, but tubercular foci, embedded in the gland, are of much rarer occurrence. Such a node appears irregular and hard at its surface, but nevertheless shows deep fluctuation. At this spot the colour of the skin is a dirty violet. The axillary glands are swollen, not hard, shotty, and small, but enlarged, ovoid, fluctuating, or even suppurating. Other signs of tuberculosis may be present.

A cold abscess, due to a tuberculous rib, situated behind the left breast, may project toward the interior of the thorax as well as toward the surface. As the deeply placed part may lie in close proximity to the heart, a distinct impulse may be communicated to the whole swelling.

CHAPTER XIV

INJURIES OF THE SHOULDER

THAT there still are physicians who are unable to recognise a simple DISLOCATION OF THE SHOULDER is incomprehensible to me. The slightest experience, and the knowledge of the normal position of the head of the humerus, compared to its position when luxated, should suffice to prevent errors.

There are other injuries of the shoulder which may cause some doubt to the beginner, for whose sake the following account is necessary. I have seen more than two hundred dislocations of the shoulder, and in more than half of these I have noticed in what fashion the beginner undertakes to make his diagnosis. I may say that in those cases in which an average student hesitates to make the diagnosis of a dislocation, it is really absent, so plain and so striking are the findings.

A skilled practitioner will at once suspect some severe injury to the shoulder when a patient enters the room with the head inclined to one side and supporting the injured arm with the healthy one. If the patient has stripped to the waist, we glance at the position of the arm and the direction of the long axis of the humerus. In the forward (preglenoid) and the downward (subglenoid) dislocation—these will be the only ones we shall discuss—the arm is abducted, and the long axis of the humerus, which normally passes through the joint in every position of the limb, will now pass to the inner side of the articulation, and, if prolonged upward, intersects the clavicle. In thus following the axis of the arm upward, our glance strikes the infraclavicular fossa. This either appears somewhat fuller than normal at its outer aspect, or a flat protrusion may be noted if the head of the humerus rests beneath the coracoid process. Observing this prominence, we examine it as a matter of course. The mass proves to be hard and rounded, and takes part in the passive movements of the upper arm. It therefore can be nothing but the head of the humerus. If the head does not appear at this spot, but is more deeply situated in the axilla (subglenoid or axillary dislocation), it is well to palpate below the acromion, pressing the finger-tips deeply inward. If the roundness of the shoulder has been lost, the dislocation can be diagnosed at a glance; but if the patient is stout, or considerable blood has extravasated, the shoulder may be as full as the healthy one. By deep palpation, as indicated above, the hollow under the acromion is demonstrable. This shows that the head has left its normal site and must now be

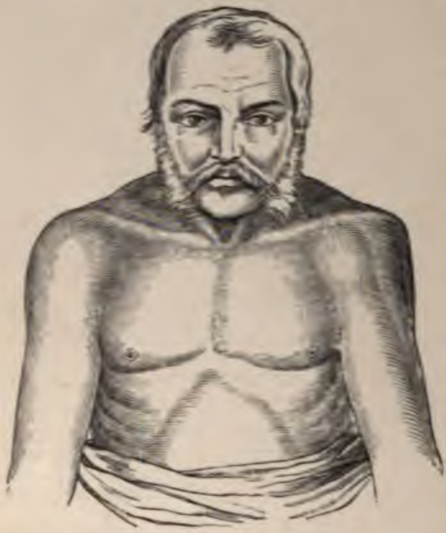

FIG. 4.

sought by examination of the axilla. If the fat or the effusion is so great that we are left in doubt, we can test for mobility. The dislocated limb is held stiffly, and attempts to bring the elbow to the trunk meet with marked resistance.

In recent cases, before effusion has taken place, and in old cases, after the swelling has disappeared, the diagnosis should be made at the first glance. The change in direction of the axis of the limb, the flattened shoulder, the flat prominence along the anterior axillary wall are so striking and so closely related, that the condition is unmistakably evident to the eye. And yet I make it a rule, in recent cases, to palpate before making a diagnosis, in order to determine one point. In some dislocations the head, after its displacement, is separated from the shaft. We must therefore test whether the head accompanies the shaft on passive movement. All signs of a dislocation may be plainly present, the above examination show that no fracture of the neck has taken place—and yet there need be no dislocation. For a fracture through the neck of the scapula sometimes occurs in such a way that the glenoid cavity forms the outer, smaller fragment. The whole arm accompanies this portion of the bone, the head remaining in the glenoid cavity, and the limb falls or comes to rest in a position which simulates a dislocation. The arm is, so to speak, dislocated upon the trunk, but it has carried its articular cavity along with it. This condition also can be diagnosed. By grasping the upper end of the arm and lifting it outward, the normal outline of the shoulder is at once restored; but as soon as the arm is released the deformity recurs. Or we may take a dislocation for granted, perform the usual manipulation for reduction, and be surprised by the ease with which it is accomplished. The deformity, however, reappears at once, and after each succeeding attempt. Frequently there is also a transverse fracture of the body of the scapula, which can not be recognised, or a fracture of the acromion. The latter can be diagnosed by obtaining a false point of motion by exerting firm pressure upon the tip of the acromion. It might be objected that this diagnosis is very incomplete, or rather not a diagnosis, for the true state of affairs is not recognised until attempts at reduction show that no dislocation is present. Can the condition be recognised earlier? If the dislocation occurs in lean subjects, the edge of the empty glenoid can be felt behind and below the spine of the scapula. Where this can be made out, fracture of the neck of the scapula may be excluded.

In the foregoing no distinction was drawn between positions of the head below the coracoid process or in the axilla. The difference is of but little practical importance, as the symptoms differ only very slightly and the methods of reduction not at all. Any one making the diagnosis of dislocation will at once be able to state which variety has to be dealt with. If the head is directly beneath the coracoid, it is a *preglenoid* or *subcoracoid*; if the head is not here, but in the axilla, the luxation is of the *infraglenoid* variety.

The rarer *intracoracoid* (subclavicular) dislocation is characterized merely by the fact that the head of the bone is to the inner side of the coracoid, below the clavicle. It can be seen and palpated. The symptoms do not differ.

The *retroglenoid* (posterior, subspinous) dislocation is extremely rare. It is marked by inward rotation of the limb and prominence of the head at the back of the shoulder below the spine of the scapula (Fig. 5).

The *luxatio erecta* of Middeldorpff can not be mistaken. The upper arm is almost vertical, and rests against the patient's head. The head of the humerus is in the axilla.

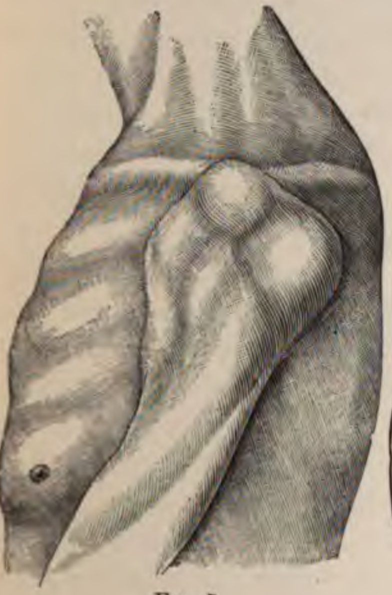
FIG. 5.

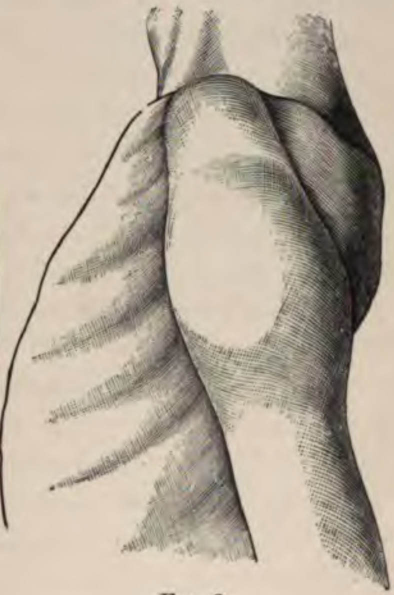
FIG. 6.

One of the rarest varieties is the *upward* dislocation. The head of the humerus lies above and between coracoid and acromion (Fig. 6).

The diagnosis is not complete without giving an account of the possible complications. Autopsy has shown that in all dislocations of the humerus a fragment is torn from the greater tuberosity. It may, however, happen that a large fragment is broken from the outer side of the head. The question then arises, whether the condition should be called a fracture with dislocation of the one fragment, or a dislocation complicated by the tearing off of a large piece of bone. The diagnosis can be made if we find a movable fragment in the region of the joint, below the acromion. Reduce the dislocation, and then call the condition anything you please.

Another complication is a simultaneous fracture of the clavicle. This can not occur until the force, which continues to act, has dislocated the arm.

Further complications are tearing or bruising of the nerves. These are diagnosed, in the later course, by the neuralgic pains, the areas of anæsthesia, and the motor palsy which they produce.

If a single symptom is alone considered, and the whole diagnosis based upon this, a dislocation may be thought of, even if the existing condition is entirely different. The flattening of the shoulder consequent to *atrophy of the deltoid* misleads many, and causes them to speak of a dislocation, or, as they hesitatingly put it, a subluxation of the shoulder. I have witnessed many such mistakes. Palpation below the acromion shows the head in place. The atrophy of the deltoid does not appear suddenly, whereas a dislocation does. The musculature covering the joint on the affected side is much thinned out.

Deviation of the axis of the humerus, as described under dislocation, will cause some to insist on the diagnosis of dislocation, even if a fracture is at the bottom of the trouble. In FRACTURES of the surgical neck, especially if the line of fracture runs from above and within downward and outward, the lower fragment assumes such a position. The shape of the shoulder is not changed, but that, as has already been said, can happen even in dislocation. A timid examiner, who fears to palpate beneath the acromion, will not know whether the head is in place. Such persons should bear in mind that the dentate edge of the lower fragment can be felt in the axilla or through the fibres of the pectoralis, and can be mistaken for nothing else. The angular outline shows that a rough fragment of bone, and not the rounded head of the humerus, pushes the soft part forward. At times the skin is almost perforated by the bone. A dislocation is naturally not thought of if the arm hangs helpless and adducted. The question then resolves itself into whether a fracture of the arm, in the vicinity of the shoulder-joint, or a simple contusion, exists. Crepitus is a symptom of much value, and the position of the crepitus—i. e., of the line of fracture—is important. If exact measurement is practicable, and no shortening has taken place, an oblique fracture of the neck is not likely, but a transverse separation is probable. This is verified by finding that the head does not accompany the shaft in its rotatory movements. This manœuvre is not always easy, and if the joint is swollen it may prove impossible. Even after the swelling has disappeared, several examinations will be needed in order to say definitely "*e pur si muove.*"

In children, ceteris paribus, the line of fracture passes through the epiphyseal line. This causes cartilaginous crepitus, and, according to Pitha, a peculiar position of the arm, with the elbow drawn somewhat backward.

The outer tuberosity may be torn away. This can be recognised, if palpation, immediately below the acromion, shows that the head moves with the shaft in rotation, but, at the same time, a part of the bone to the outside of the joint and below the acromion does not accompany movements of the shaft. Active inward rotation is possible, but outward rotation is not.

In old people the insertions of the external rotators alone may be torn from the tuberosity. The function is disturbed as above, and only a small, movable fragment of bone can be felt.

More detailed directions for examination can not be given. At the time that a swelling obscures the joint, dislocation, at least, must not escape notice. In suspected fractures a more accurate diagnosis may be left *in suspenso*; it will not harm the patient. Certain forms of fracture it is impossible to recognise. Gurlt mentions rare cases in which fragments were broken from the head within the joint, and one case in which the head had separated and turned so that its cartilaginous surface was in contact with the lower fragment.

Fracture of the clavicle can ordinarily not be overlooked. Even if the characteristic inward and forward droop of the shoulder were not recognised, the line of fracture on the subcutaneous clavicle can be palpated; and if more than one fragment has to be dealt with, this also is evident. In children, in whom transverse subperiosteal fractures occur, and in fractures close to the acromial end of the clavicle, the line of fracture can not be distinguished. In these cases, in addition to local pain on pressure (often slight ecchymoses), pain is elicited at the same spot by movements of the arm or of the clavicle, if this is grasped at the sternum and moved. Moreover, the arm can not be actively raised.

I have seen beginners in doubt between a *dislocation* *of the clavicle at its acromial end* and fracture, when the first condition had to be dealt with. This hesitation is partially justified if the acromial end is dislocated upward and, as is always the case, backward. Measurement will show that the distance from the edge of the sternum to the acromial end of the clavicle, which projects under the tightly stretched skin, corresponds exactly to that of the opposite side. Palpation will show that the projecting bone is not sharp and serrated. Dislocation *beneath* the acromion is harder to distinguish. Careful palpation is needed. The acromion is found to project boldly at its inner side, and the clavicle is lost beneath the projection. Fractures of the coracoid process of the scapula must be diagnosed by abnormal mobility and crepitus. Nothing further can be added.

Hochenegg has called attention to an interesting fact. When the long (scapular) tendon of the biceps is torn through above, the muscle contracts down upon itself, its belly shortens, and the upper region appears flatter and emptier than normal.

CHAPTER XV

INFLAMMATIONS AND TUMOURS IN THE VICINITY OF THE SHOULDER-JOINT

It is of paramount importance, in inflammatory processes occurring about a joint, to determine whether the inflammation is taking place *within* or *without* the joint cavity. As the tissues which surround the shoulder-joint and form its support are of manifold kind, a variety of extra-articular or periarticular swellings are possible. Abscess of a muscle is rare as a primary complaint; therefore the muscles are not among those structures which, by their inflammation, give rise to periarticular abscess. Between the capsule and the muscles are *bursæ*; they and the cellular tissue, found on the outside of the capsule, give rise to periarticular abscesses upon suppuration. The bursæ which must be considered are the subdeltoid, the subacromial, and the subscapular, of which the last may be regarded as a diverticulum of the capsule because of its constant communication with the joint cavity. A second diverticulum is formed by the bursa, which accompanies the long tendon of the biceps along the bicipital groove. In addition, Gruber has described a number of small bursæ about the coracoid process, which not infrequently are affected. Acute and chronic serous effusions take place, and, by preference, suppurative processes. More frequently tubercular caries occurs at the upper end of the humerus, the pus pointing extra-articularly beneath the deltoid. The resulting cold abscess will present the symptoms of a subdeltoid bursitis.

Exudation into the subdeltoid bursa, or a cold abscess beneath that muscle, makes the deltoid more prominent. Effusion into the subacromial bursa causes a small swelling, which can be seen and felt directly below the acromion. Effusion into the subscapular bursa, not communicating with the joint, produces a swelling in the axillary region which is best seen when the arm is raised. It would not seem improbable that an effusion into the large joint cavity would give a more complicated picture than effusions limited to smaller regions. And yet, as a matter of fact, this is not strictly true. Serous or purulent effusion into the subdeltoid bursa, or a cold abscess in this neighbourhood, changes the outline of the shoulder more perceptibly than does a considerable effusion into the joint itself. A collection of fluid beneath the deltoid raises the entire muscle from the capsule and increases the size and roundness of the shoulder to a marked degree (Fig. 7). Fluid in the joint never bulges out the deltoid region, because the strong re-enforcing band of the capsule, which is situated here, does not yield. The capsule first bulges posteriorly, fluctuation being most readily demonstrated behind, below the spine of the scapula. It is at this same spot that tuberculosis of the shoulder-joint is most apt to come to the surface. Prominence of the deltoid region is misleading, and if not corroborated by other symptoms, is insufficient to confirm the diagnosis of arthritis.

As in other joints, we would expect to find a pathognomonic position. Experimental and anatomical data would point to abduction, rotation outward, and a slight forward inclination of the arm as the position of rest. Clinical experience teaches that this position of rest is not found in inflammations of the shoulder-joint, even if considerable fluid has accumulated. I have seen it at the onset of severe traumatic inflammations, but masked by the inclination of the body—i. e., concavity of the spinal column directed toward the injured side. In the dependent position of the arm the fibres of the capsule are not twisted, but merely somewhat folded upon each other, so that this posture is borne as well, and more often found, than the experimentally determined position of rest.

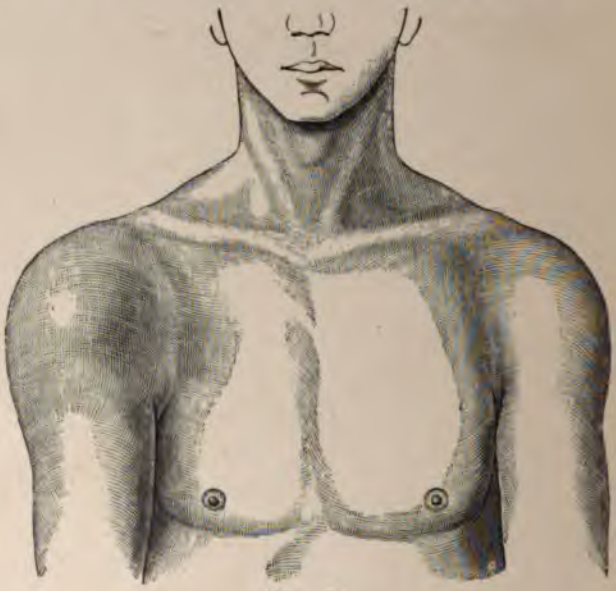

Fig. 7.

As a pathognomonic position does not occur, the diagnosis of an inflammation of the shoulder-joint is based upon another sign. In a single word, it is the *fixation* of the joint. Just as the pelvis and thigh are held in a certain fixed position in inflammations of the hip-joint, the humerus and scapula are fixed in a definite position to each other. In coxitis the pelvis is said to move with the thigh, and in inflammations of the shoulder-joint *the scapula moves with the arm.*

In practice this is the first sign that is looked for. Place the fingers of one hand upon the shoulder-blade, with the other hand grasp the arm, and attempt to make it describe various movements. If the scapula moves with the limb in every direction, the underlying condition is an inflammation of the joint. The scapula normally takes part in certain movements. This is especially the case in movements in a sagittal and frontal plane after the arm is raised above the horizontal. These movements never take place without participation of the scapula, and some slight participation is apt to occur even below the horizontal. But with the scapula fixed, rotatory, sagittal, and slight movements in the frontal plane can be made without accompanying movement of the scapula.

In inflammatory processes in the vicinity of the joint, movement of the arm is somewhat limited in those directions which cause pain, but not in movements which may be painlessly executed. But if mobility is entirely abolished, a joint process must be the cause (anchylosis is included as a final outcome of arthritis). This, therefore, is the chief symptom.

Immobility of the shoulder-joint materially decreases the range of movement of the arm, but with the aid of the scapula the upper extremity still enjoys considerable mobility. A certain amount of abduction, some elevation in the sagittal plane, and a slight degree of rotation, are still permitted. Greater elevation of the arm is possible by a combination of abduction with elevation in the frontal plane. These movements take place in the sternoclavicular joint of the diseased side, and are therefore limited by the range of mobility permitted by this articulation. In chronic, almost painless inflammations, usually the result of tubercular caries, the sternoclavicular articulation is called upon to perform more than its usual share of work, because, in spite of the caries, many movements can be performed without discomfort. In such cases the range of motion enjoyed by the joint becomes greater, and the clavicle stands out more prominently from the sternal articulation. This is a symptom regularly noticed in this variety of inflammation.

In the associated movement of the scapula we have an important symptom, the first one to be employed in differential diagnosis of inflammations in this region. In those cases in which there is a fluctuating swelling, the next question to decide is whether the fluid is extra-articular or intra-articular. A large intracapsular effusion can be felt posteriorly below the spine of the scapula, and internally in the axilla. Medium-sized exudates can be felt only behind, where suppurative processes likewise point. An acute effusion in the joint never causes the deltoid to bulge out; this can alone occur in chronic conditions.

Such marked distention of the capsule is, however, of rare occurrence. Tuberculous disease of the shoulder-joint with fungous degeneration of the capsule, which might give the signs of joint effusion, is also remarkably rare. In the shoulder-joint, most inflammations take place unaccompanied by much swelling. As a rule, therefore, the inflammation is only recognised by the *complete fixation of the joint, and pain throughout its entire extent.*

In practice, large effusions into the joint are caused by:

1. Suppuration following open wounds penetrating the joint (sword and gunshot wounds).

2. Rupture of pus into the joint (especially in osteomyelitis and more rarely in deep cellulitis).

In these cases the fixation of the joint is the dominant symptom. As a rule, however, the whole neighbourhood is swollen, because not only the joint cavity, but also the adjacent supporting structures, are involved in the suppurative process.

If the suppuration continues for some length of time, sinuses leading in various directions may form. The cartilaginous surfaces are eroded, so that a rough crepitus is elicited by movement of the joint. The process may finally lead to extensive destruction of the capsule, and consequent abnormal mobility of the humerus within the glenoid cavity.

When the suppuration has healed, anchylosis of the joint results. Old cases of anchylosis at the shoulder-joint may be due to the following causes:

1. A healed traumatic suppurative arthritis.

2. A healed osteomyelitis of the upper end of the humerus.

3. A healed tubercular arthritis or synovitis.

4. A healed suppurative arthritis due to rupture of a deep phlegmonous focus into the joint.

At least one of these diseases is strongly characterized, namely, *osteomyelitis.* An acute initial disease, resembling typhoid, occurring during the period of growth; multiple abscesses, and, eventually, discharge of sequestra spontaneously or by operation. Fistulæ, leading down to loose bone or scars attached to bone, have the same significance. *Thickening and deformity of the upper end of the humerus*, lengthening of the bone, and, as a final outcome, marked shortening.

These last-mentioned bone symptoms differentiate the condition from deep cellulitic inflammations.

Tuberculosis has no acute initial stage, no lengthening or thickening of the bone. The tubercular habitus and other local tubercular symptoms can usually be demonstrated. One variety—*Caries sicca*—is peculiar to the shoulder-joint. Complete immobility *without* swelling, but on the contrary with wasting of the muscles, shortening of the bone (due to destruction of the head), and severe pain, characterizes this process.

Neuropathic disease of the shoulder-joint was formerly unrecognised; to-day it is often wrongly interpreted, although it is not difficult to distinguish. It is a chronic, deforming arthritis marked by great effusion into, and distention of, the capsule. The whole region is markedly swollen, and the capsule is weakened. The head is finally subluxated, and loud crepitus readily elicited. Formerly this symptom-complex sufficed to establish diagnosis of arthritis deformans.

Bearing trophic joint diseases in mind, each case should be examined for *tabes* and *syringomyelia*, and the patient tested for dissociation of sensation or trophic disturbances of various kinds. A joint trouble surprisingly often leads to the discovery of some central nervous disease.

Axillary abscesses are of importance in this connection because the more deep-seated ones are in close relation to the structures of the shoulder. Just as in the hip-joint, periarticular inflammations may lead to fixation of the joint, and result in partial anchylosis. A still more important outcome, which has been previously referred to, is rupture of the abscess into the joint.

Velpeau classifies abscesses in the axilla into superficial or nodular abscess of the cellular tissue, phlegmonous erysipelas (suppuration of the subcutaneous tissue), suppurative lymphadenitis, and deep cellulitis.

The first variety is seen in the form of several small abscesses which appear in the hairy skin of the axilla as nodules, reaching the size of a pea or bean. Inflammation of the lymphatic glands is recognised by the occurrence of circumscribed nodular swellings, with isolated harder spots in the periphery. These harder portions represent the glands which have not yet broken down. Both varieties are characterized by the circumscribed form of the swelling. Superficial and deep cellulitis are *diffuse* processes, which may be distributed simultaneously on the inner side of the arms, the side of the breast, and the shoulder. It is only necessary to decide whether the process is superficial or deep-seated. As far as treatment is concerned, this is of little importance, as early incision is indicated in both; but from a prognostic standpoint the distinction is important. The deep axillary abscesses are notorious. The son of the famous J. L. Petit died as the result of such a process, and even the more ancient writers mention the destruction caused by the far and wide burrowing of the pus. Velpeau describes the directions taken by such abscesses. They may follow the course of the large vessels, and proceed along the brachial plexus, either above or below the clavicle, burrow backward between the latissimus dorsi, the trapezius, the rhomboideus, and the serratus magnus; some spread in all three of these directions. When they point into the mediastinum or penetrate the pleura a fatal outcome is the rule. The fasciæ are considered as boundaries between superficial and deep axillary abscesses; the diagnosis of the two conditions is, therefore, made just as elsewhere in the body. Severe constitutional symptoms, œdema in the neighbourhood, late appearance of fluctuation, in spite of great swelling and tension of the parts, may be regarded as the marks of a deep suppurative process. Symptoms distinctive of this region also appear; these have already been indicated. If tension, and later fluctuation, can be demonstrated beneath the clavipectoral fascia, in the subclavian triangle, or in the supraspinous fossa, the abscess is surely deep-seated. Burrowing along the muscles of the arm occurs, but is rare.

Finally, I may refer to the so-called *scapular crepitus*. At times a loud, rough crepitus, accompanied by pain, will be heard at the lower border of the scapula on movement, although the shoulder-joint is intact. It is caused by contact of the scapula with the ribs, due to a chronic inflammation which has resulted in destruction of the interposing muscles and roughening of the bony surfaces.

CHAPTER XVI

DISLOCATIONS AND FRACTURES AT THE ELBOW

SUBCUTANEOUS injuries to the elbow-joint and the bones that aid in forming the articulation are both very numerous and of diagnostic interest. The following dislocations are found: both bones of the forearm may be dislocated, backward, outward, inward, or forward, either together or in opposite directions (as, radius forward, ulna backward—divergent dislocations). Both may be dislocated forward, but complicated by torsion, so that the concavity of the coronoid process of the ulna faces backward in moderate extension of the forearm (reversed dislocation—*Umkehrungsluxation*). To these may be added incomplete dislocation of both bones inward and outward, which swells the total to eight varieties. The radius may be dislocated in three directions—outward, backward, and forward. The ulna alone may be displaced backward. These increase the possible number of dislocations to twelve. The commonest of all is a backward dislocation of both bones; the luxations of the radius alone are also not infrequent, and incomplete lateral dislocations occur with greater frequency in children than is generally supposed. The other forms are rare.

Various fractures require consideration from the standpoint of differential diagnosis. Fracture of the lower end of the humerus is common, and resembles backward dislocation of the bones of the forearm. Other fractures are fracture of the condyles of the humerus, of the coronoid process of the ulna, of the upper end of the radius, and, lastly, in children, epiphyseal separation of the lower end of the humerus.

Incidentally fractures of the olecranon may be mentioned. They rarely give rise to difficulty in diagnosis.

Let us consider the more common varieties referred to above. A patient presents himself, suffering from an injury to the elbow-joint sustained by a fall from a wagon. The whole region is occupied by a considerable swelling, which obscures the contour and extends up the arm and down the forearm. After the patient has stripped to the waist, we measure the length of the limb. This is shortened about two centimetres, if roughly gauged by sight. The joint is slightly flexed, about one fourth of the possible degree of flexion. I can not sufficiently reprove the fault common to beginners, of at once grasping, handling, and pressing an injured part without rhyme or reason. I will show how a purposeful inspection reduces this handling to a minimum. Inspect as long as further information can be gained by the eye. Of great importance is recognition of the relative position of the axis of the forearm to that of the arm. In our patient we notice that the axis of the forearm has been moved backward, though still parallel to its former course. Draw an obtuse angle (⌟) in which the vertical line represents the axis of the arm, the lower, almost horizontal line, the axis of the forearm. If, now, the vertical line is left as before, but the lower line is moved backward, parallel to its former position (⊥), the existing condition is diagrammatically reproduced. Moreover, the axis of the arm and of the forearm no longer intersect at the joint, but at a point situated on the forearm. This is due to the fact that the forearm slips upward at the time of dislocation. For the same reason, the forearm appears shortened, especially if examined from in front, but the upper arm must also appear shortened along its extensor or posterior surface. All these findings confirm the same fact, merely regarded from different standpoints. The upward displacement of the forearm accounts for the actual shortening, which does not exceed one or two centimetres. Inspection would lead us to conclude that the case is one of *dislocation.* In transverse fracture of the lower end of the hu-

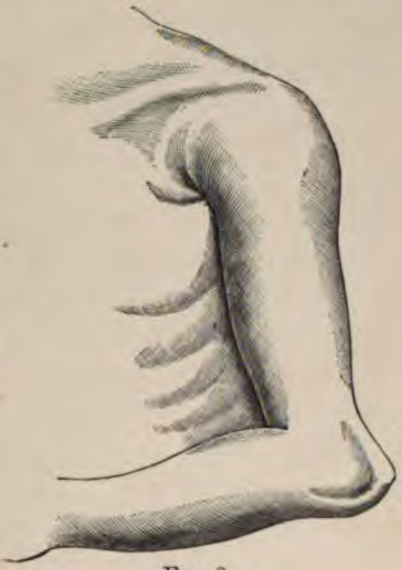

Fig. 8.

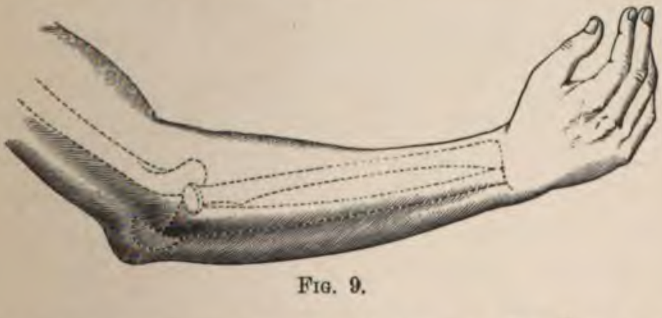

Fig. 9.

merus, the forearm, and the lower fragment can assume the above position, so that all the previously mentioned symptoms—shortening, position, and axial deviation—are common to both. A single manipulation is sufficient to decide. Grasp the forearm and partially flex it. If the resistance encountered on attempting flexion is insurmountable, dislocation and not fracture has to be dealt with. Should a sceptical colleague refuse to be convinced, still further evidence may be advanced. Find the internal epicondyle, which is readily located no matter how extensive the swelling, and then the less prominent external epicondyle. With the elbow at a right angle, the two epicondyles and the tip of the olecranon should normally lie in the same straight line. In dislocation, the tip of the olecranon is as much above this line as the forearm is shortened—in adults, about two centimetres. If this symptom is to be clearly demonstrated, we stand in front of the patient and flex both elbow-joints to a right angle in order to compare the healthy and the injured side. The middle finger rests against the internal condyle, the thumb against the external, and the index-finger upon the tip of the olecranon. On the sound side the three fingers are in line; on the injured, they represent the corners of a triangle, of which the index-finger is at the apex. This, however, merely proves that the ulna has been dislocated. The radius must also be accounted for. Normally, the head of the radius is about a finger-breadth below the external epicondyle. In dislocation this space is empty, and the depression found on the head of the radius can be felt behind the condyle. Passive pronation and supination serves to identify the head, which is felt to accompany the shaft in these movements.

In *fracture*, the forearm can be flexed beyond a right angle, extended beyond a straight angle. The relation of the two epicondyles to the olecranon are unchanged; the head of the radius is in its proper place. If we grasp the two condyles—i. e., the lower fragment—it can be moved to and fro, as the point of abnormal mobility is above the epicondyles. A T-shaped fracture should not be overlooked. The transverse fracture, which separates the lower end of the humerus from the shaft, is then complicated by a vertical fracture, which divides the lower fragment into two pieces. In this case the condyles will move on each other, or crepitus limited to the lower fragment may be elicited.

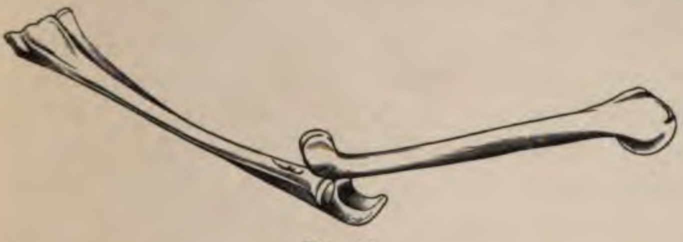

FIG. 10.

The picture of a recent dislocation without swelling, or of an old dislocation, is so striking that it can never be forgotten after one case has been examined. It can be produced on the cadaver by overextension, followed by flexion, combined with strong backward pressure exerted upon the forearm. The symptoms of this dislocation, described by all authors, are then plainly evident. The flexor surface of the forearm and the extensor surface of the upper arm seem shortened. The triceps tendon forms a marked curve with concavity directed backward, and the olecranon becomes unduly prominent with depressions on either side of it. The head of the radius allows the posterior half of its articular surface to appear, while anteriorly the trochlea, especially the mesial edge, can be felt. The transverse diameter of the joint is unchanged, etc.

It might be objected that dislocation of both bones of the forearm, or fracture of the lower end of the humerus, are not the sole conditions to be dealt with. Therefore, while resistance to flexion beyond a right angle is a striking symptom, it may be of no value in the diagnosis of injuries to the joint.

Let us analyze the objection: *Lateral* dislocation of both radius and ulna can be recognised at the first glance. The forearm has left its normal site, and has moved either to the inner or outer side. The axis of the humerus and of the forearm run side by side; this relation is diagrammatically represented as follows: └┐. This variety requires no further discussion, as it can not be confused with the preceding forms. *Forward* dislocation of both bones is at once excluded by the position of the forearm. In this luxation the elbow is held rigidly at an acute angle of flexion. The so-called reversed dislocation attracts attention because of the marked pronation. The protuberance noticed at the front of the joint proves to be the subcutaneous surface of the olecranon, and the site normally occupied by the olecranon is empty.

Divergent dislocations are characterized by the considerable degree of shortening, combined with readily recognised separation of radius and ulna at their upper ends.

In dislocation of the *ulna alone* (Fig. 11), the forearm assumes a pathognomonic position. It is fixed in complete extension. The axes of the arm and forearm form an obtuse angle, with its apex directed away from the median line, resembling somewhat the lower extremity in genu varum.

All these varieties differ widely from the first two conditions mentioned, and, as a matter of course, lead to different deductions. This leaves only dislocations of the radius and incomplete dislocations of the forearm to be considered. Dislocation of the radius causes a swelling, which is specially prominent at the radial side. *Posterior* dislocation of the radius limits flexion beyond a right angle, and is thus differentiated from fracture of the lower end of the humerus. It can not be confused with dislocation of both bones backward, because no shortening takes place. *Forward* and *outward* dislocation of the radius in some cases impedes movement and in others does not; but, as in backward displacements, if measurements are taken along the ulnar side of the forearm, shortening is absent.

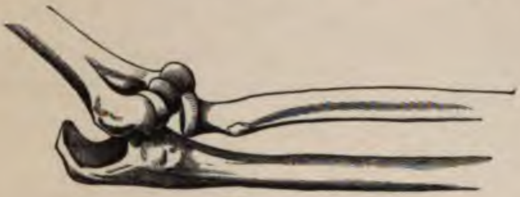

FIG. 11.

Incomplete dislocations of the forearm still remain to be discussed. They offer considerable difficulty in their diagnosis, as is shown by the greater frequency with which they are recognised in recent days. Streubel's classical work on dislocations of the elbow, which appeared about thirty years ago, mentioned only a few scattered instances, while Pitha, in his treatise on surgical diseases of the extremities, records three cases of his own. Hüter, alone, saw eight cases, most of which were of old standing, unreduced, and probably unrecognised. At v. Dumreicher's clinic I saw three cases, and since then I have seen many more. It is now generally known that these dislocations, formerly unrecognised, frequently occur in children. The dislocation results either in an outward or an inward displacement; that is, toward the radial or ulnar side. Of these, *outward* displacement is by far the more frequent. The position of the bones of the forearm is as follows: the radius is displaced outward from the eminentia capitata, and rests beneath the external epicondyle without producing a marked prominence. The ulna moves with the radius, so that the centre of the sigmoid cavity no longer corresponds to the centre of the trochlea. In almost every case the internal lateral ligament has torn away part of the internal epicondyle. It is just this small fractured surface, felt upon the internal condyle, which is misleading. The first case I saw caused me some misgivings. The joint was obscured by swelling. Shortening was absent; flexion beyond a right angle unimpeded; in fact, the range of flexion was greater upon the diseased than upon the sound side. This definitely excluded backward dislocation of both bones of the forearm, but not fracture of the lower end of the humerus. In isolated cases flexion has been found impossible. In these, absence of shortening still suffices

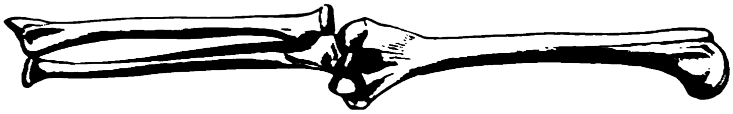

FIG. 12.—Right arm.

to exclude backward dislocation. In my case I suspected fracture of the lower end of the humerus, as I felt a sharp fragment of bone. How was the lack of shortening to be accounted for? Transverse or slightly oblique fractures at the lower end of the humerus may occur without shortening. I have seen a few in which the line of fracture traversed the internal epicondyle. Displacement is then not necessarily *ad longitudinem*, but rather *ad latus*. The lower fragment accompanies the forearm outward; the lower end of the upper fragment protrudes inward. This lateral displacement produces a relation of the axes of the limb like that seen in incomplete outward dislocation. The resemblance of the signs, on inspection, is great; palpation alone can decide. In incomplete dislocation we recognise by palpation the inner half of the trochlea, either below the fractured edge of the base of the epicondyle, if this has been torn away, or beneath the epicondyle itself, if this has not been fractured.

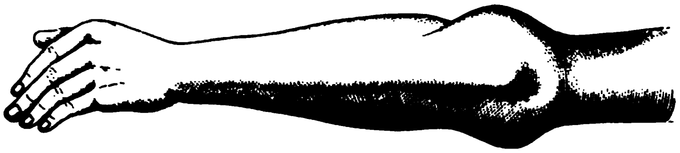

Fig. 13.—Left arm from without.

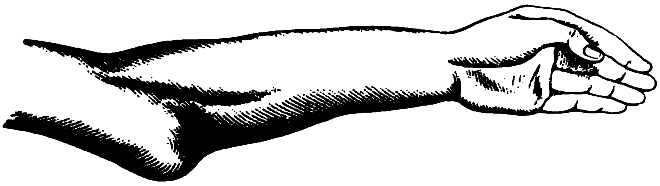

Fig. 14.—Left arm from within.

Even if the swelling is extensive, the steep inner edge of the trochlea can be plainly felt, and, what is even more important, is found immovable. Beneath it is a hollow, which opposes no resistance to pressure because it is no longer occupied by the coronoid process. The very fact that the piece of bone felt *below* the line of fracture is *immovable* shows the fallacy of assuming that a fracture of the lower end of the humerus has occurred, and further palpation shows that this fixed piece is the empty trochlea. Such was my experience.

In *incomplete* dislocations toward the *ulnar side*, the articular surface of the coronoid process has surmounted the sharp internal edge of the trochlea; the radius has left the capitellum, and lies in contact with the trochlea, deeply hidden by the soft parts. Shortening is absent and flexion unimpeded. The characteristic symptom is the exposed portion of the internal edge of the sigmoid fossa of the ulna, which can be felt beneath the internal epicondyle (Fig. 15).

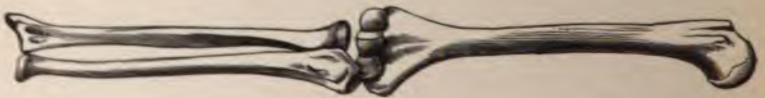

FIG. 15.—Right arm.

We might say that by emphasizing these characteristic signs, other symptoms being taken for granted, palpation below the internal epicondyle will suffice to show whether an incomplete outward or inward dislocation has taken place. In outward dislocation the trochlea can be felt; in inward displacement, the fossa sigmoidea. The free border of the trochlea forms an edge *convex* anteriorly; the free border of the sigmoid, an edge *concave* anteriorly. A study of the bones entering into the formation of the elbow-joint will illustrate these remarks.

Fluoroscopic examination of both recent and old cases tends to prove that these dislocations rarely occur in children in an *uncomplicated form*. As a rule, some fracture is present, so that the condition may be called a fracture-dislocation. We have already discovered a great variety of combinations. In one case the ulna was displaced downward (distally). Our knowledge of these conditions will surely be increased by further observations.

As has been previously stated, backward dislocation of both bones of the forearm, although of frequent occurrence, is an often unrecognised injury. Two other conditions which bear some resemblance to it have not yet been discussed.

In children, *separation of the lower epiphysis of the humerus* takes place. The line of separation runs below the epicondyles; the lower fragment consists only of trochlea and capitellum. It is evident that the forearm, in company with the lower fragment, can be displaced backward. The olecranon is then prominent posteriorly, and is displaced upward; the radius has left its normal position, and the forearm is shortened. The symptom-complex resembles that produced by a dislocation. Palpation discloses the tip of the olecranon above a line drawn between the epicondyles, and as the relative position of the olecranon to the epicondyles is altered, we are led to assume that a dislocation exists. But, on testing flexion beyond a right angle, no resistance is encountered. This seeming incongruity—viz., relation of the anatomical points as in dislocation—combined with unimpaired flexion, is characteristic of epiphyseal separation.

In other instances, the opposite displacement, of both forearm and lower fragment, occurs, namely, displacement *forward*. The olecranon has left the back of the joint; the line of separation passes below the epicondyles. Correction of the deformity is very easy.

The second form of injury, which resembles dislocation, is *fracture of the coronoid process of the ulna*. But few cases are on record; I have seen one case myself. In no instance was this injury found alone, some other complication always existed, although the fracture of the coronoid was the most noticeable feature. The striking symptom is the prominence of the olecranon, which is found above the intercondyloid line. If this injury is thought of, palpation beneath the external epicondyle shows that the radius is not displaced. Dislocation backward of the ulna, alone, will then account for the symptoms. As a matter of fact, the ulna is dislocated because the coronoid process, which grasps the trochlea, has been broken off. Compared to uncomplicated dislocation of the ulna, there is some difference in the position of the forearm, which is more adducted (ulnar adduction) in simple dislocation. If the torn and often movable coronoid process can be palpated in the bicipital fossa, it would be conclusive; but, as Pitha remarks, extreme delicacy of touch is required

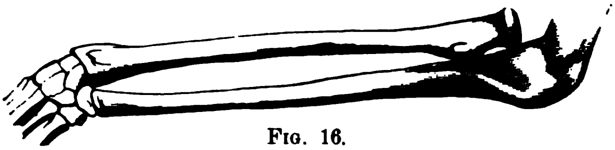

FIG. 16.

to elicit this symptom. Another symptom, however, is quite as valuable. By downward traction of the ulna the olecranon grows less prominent, but as soon as the traction is discontinued, and slight attempts at flexion are made, the deformity promptly recurs.

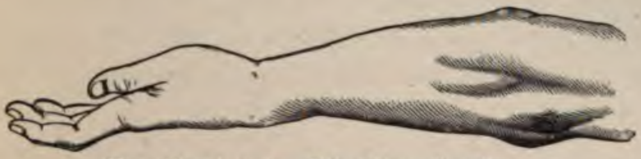

FIG. 17.—Anterior dislocation of radius.

Dislocations of the *radius alone* demand further mention. Absence of shortening distinguishes them from dislocation of both bones of the forearm, even if the extent of the swelling obscures the joint and the dislocation of the radius hinders flexion beyond a right angle (by no means a constant occurrence). If the swelling is great, careful palpation of the outer side of the joint must be practised; for, if felt, the head of the radius can not be mistaken for any other structure. In recent cases it may be seen protruding beneath the skin (in external and posterior dislocations), or the contour of the external condyle is abnormally distinct (forward dislocation). One point, however, may be overlooked: this is fracture of the ulna, especially of the middle third, which often accompanies dislocation of the head of the radius. The fracture may escape unnoticed or may catch our eye, while the dislocation escapes observation. By remembering that these injuries often occur together, mistakes will be avoided.

Fracture of either condyle of the humerus causes no pathognomonic posture, no shortening, and no limitation of motion. In these cases careful palpation is an essential to correct diagnosis. The same holds true of *fracture of the head of the radius*, for absence of rotation of the head in pronation and supination is a very deceptive symptom, especially if the dislocation is slight. As a rule, displacement is marked, and the upper fragment lies almost transversely with its sharp fractured end toward the ulna.

Let us now briefly review the question of *fracture versus dislocation*. The preceding paragraphs have shown that several of these injuries are to be recognised at the first glance, while others require careful examination. In this examination, the discovery of an exposed joint surface or of a protruding process—i. e., recognition of the head of the radius, the edge of the sigmoid fossa of the ulna, exposed trochlea, or capitellum—prove that a dislocation has occurred. In determining whether the commonest variety—namely, backward dislocation of both bones of the forearm—has taken place or not, the depression on the head of the radius can always be felt, no matter how great the swelling, and the sigmoid fossa is found empty, even if its whole articular surface can not be explored.

Finally, there is a condition which can be demonstrated with the aid of the X-ray. This is the ossification of isolated structures in the neighbourhood, after fracture in the vicinity of the elbow-joint. For instance, the insertion of the brachialis anticus or the biceps may become ossified. Formerly these were mistaken for bony fragments.

CHAPTER XVII

INFLAMMATORY PROCESSES OF THE ELBOW

WHEN the elbow-joint is distended by a fluid exudate, it assumes a position of flexion at an angle of about 120°. The anteroposterior diameter, measured from the crease of the elbow to the tip of the olecranon, is the only one which is noticeably increased. This is due to the fluid which raises the anterior part of the capsule from the trochlea and capitellum. The most marked changes in outline take place posteriorly. Normally furrows are to be seen on either side of the olecranon; these are now not only obliterated, but replaced by swellings. The capsule in this situation is thin and not tense; it can, therefore, be distended by the effusion, and in chronic cases is stretched. A sharp eye will notice that the bony outline in the vicinity of the radio-humeral articulation (a finger's breadth below the external epicondyle) is masked by the effusion which surrounds the head of the radius. All these signs can be studied on the cadaver by boring through the olecranon, and distending the joint through this opening by means of a cannula.

The *fluid* nature of the exudate is recognised by the fluctuation, which can be felt in the swellings found on either side of the olecranon. In those cases in which the effusion is considerable, direct communication between the two may be demonstrated by pressing upon one swelling and finding that the other grows more tense. This is proof positive that the effusion is in the joint, because the sole communication must be anterior to the olecranon, and the anterior surface of this process helps to form part of the joint cavity.

This is the ordinary picture of a SEROUS EFFUSION due to trauma or rheumatism. In cases in which a tubercular habitus, heredity, or tubercular foci elsewhere, attract our attention, we must not forget that a *serous tubercular synovitis* is not an impossibility, even as a sequel to trauma. The prognosis must accordingly be guarded. The further course of the trouble will decide, as other more characteristic signs of joint tuberculosis are sure to appear. If circumscribed spots, tender to pressure, develop early in the bones, it should be regarded as an ominous sign, which points to tubercular foci.

TUBERCULOSIS of the elbow-joint does not, however, usually present the picture of a fluid exudate, but rather that of a proliferation of the capsular tissues (fungus). The symptoms are identical with those mentioned above, except for the *absence* of fluctuation in the swellings on either side of the olecranon. The joint appears to be full, but the contents is not fluid; it follows that the capsule must be thickened by the development of new tissue—the soft, elastic tissue of fungus, as it was formerly called.

As a rule, patients do not seek advice until the disease has reached a more advanced stage. The extensor surface of the joint is found spherically rounded; the olecranon with its two welts has disappeared in the general change in contour, which has obliterated all the landmarks. The skin has a dull lustre, and the swelling is doughy to the touch. At this time some lateral mobility may already be present, and consequently the patient supports the affected arm.

The process is even more strikingly developed if the swelling has become *spindle-shaped*, and merges into the tissues of the arm and forearm. The bone has been widely attacked, the ligaments destroyed, and crepitus, if present, shows that the cartilaginous surfaces have become eroded. Sharply circumscribed spots of fluctuation point to breaking down of part of the fungous mass, or, in more advanced cases, such abscesses have pointed and discharged. They leave a characteristic tubercular ulcer, with thin, sharp, undermined violet edges. In children, this picture may develop with great rapidity.

When tuberculosis of the elbow-joint begins in the bones forming the articulation, a cold abscess is apt to develop early within the area of general infiltration. The position of the abscess often indicates the site of the bony focus. This is particularly true of abscesses on the back of the olecranon, which positively point to a tubercular focus in this bony prominence. The outer side of the lower end of the humerus is more often affected than the inner, and not infrequently the head of the radius is the starting-point of the disease.

Occasionally tubercular arthritis of the elbow-joint runs a very chronic course. After one or two years, nothing more than non-fluctuating swellings to either side of the olecranon and limited range of motion may be found. If the process has developed spontaneously, only one of two conditions need be considered—tuberculosis and *arthritis deformans.* High degree of tension in the swellings to the side of the olecranon, marked grating crepitus, tumefaction of the head of the radius, hard, uneven bodies at the insertion of the capsule posteriorly, speak in favour of arthritis deformans.

In acute inflammations of the elbow-joint a differential diagnosis between articular rheumatism and *diffuse osteomyelitis* of the lower end of the humerus may be difficult. The condition is more obscure if, in addition to a periarticular inflammation, an intra-articular effusion has occurred, for osteomyelitis occasionally begins with an effusion into a neighbouring joint cavity. Since we know that osteomyelitis is not limited to patients below the age of twenty-five, more advanced age does not exclude the presence of this affection. Doubt can only last a short time, for as soon as evidence of suppuration is noticed, rheumatism is out of the question. Even previous to this, œdema spreading over the whole forearm and great sensitiveness of the bone to pressure favour osteomyelitis. If elsewhere in the body bony swelling appears at some distance from a joint, the case is clearly one of multiple osteomyelitis.

It must be kept in mind that *trophic* disease of the elbow-joint occurs, but does not follow the type of an arthritis deformans. In all cases in which the picture does not agree with the tubercular or other typical forms, some lesion of the central nervous system (even in young subjects) should be suspected.

Inflammatory *extra-articular swellings* of this region require but little discussion. Inflammation of the *olecranon bursa* is readily recognised by the circumscribed fluctuation, which is exactly limited to the extent of the bursa. This process may become serious, as it frequently shows a marked tendency to suppuration and rapid extension down the forearm. *Chronic bursitis* can not be mistaken; the grating, due to the excrescences on the surface of the bursa and to the free rice bodies, is characteristic. Contusions at the back of the elbow may cause a *hæmatoma* of the bursa. The clotting blood often gives rise to a soft, fine fibrin crepitus.

Acute and chronic abscesses may be found at the lower end of the *bicipital sulcus* along the inner side of the joint. They will pass unrecognised by those who are ignorant of the position of the cubital glands. The abscess may reach the size of a hen's egg, but never endangers the joint.

In the course of general metastases, abscesses may develop outside the capsule, especially on the flexor surface. If, in a patient suffering with pyæmia, the joint cavity is uninfected, the sudden pain, the slight swelling over the flexors, with absence of swelling posteriorly, will point to the diagnosis.

CHAPTER XVIII

INJURIES OF THE HAND

The great number of small articulations in the carpus increases the number of pathological processes usually met with. The interesting diseases of the synovial tendon-sheaths, which surround the wrist and extend into the fingers, also require mention. Injuries of the thumb, especially the notorious dislocation of its first phalanx, almost deserve a separate chapter, while Dupuytren's contraction still further swells the list of affections.

French neurologists, in describing the posture and shape of the hand so characteristic of palsy of the main nerve-trunks, have applied the names ordinarily used

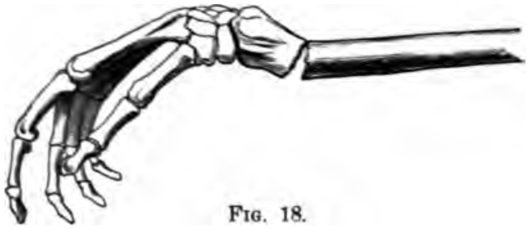

Fig. 18.

in the description of physiognomy. They speak, for instance, of the hand of the pathetic preacher. The surgeon is reduced to the plainest prose—he studies the abscess of the hand, hidden beneath the horny palm of the cobbler or carpenter; he cures the panaritium of washerwomen and servant-girls, and puts an end to their sleepless nights by incisions, the depth of which bear witness to his diagnostic skill.

FRACTURE OF THE LOWER END OF THE RADIUS deserves our first attention. Direct violence may fracture the bone in any situation, but indirect, as a fall upon the outstretched hand, causes a fracture immediately above the wrist. The injury is the result of tearing rather than of breaking of the bone. A fall upon the dorsally flexed hand puts the ligaments upon the stretch; not they, but the lower end of the radius, give way, so that the carpus and the lower fragment remain united. This fracture is frequently overlooked by ignorant practitioners. Pitha puts it too mildly when he says, "It is often difficult to make physicians realize that a fracture has occurred." The symptoms are by no means insignificant. As soon as the limb is fractured, the upper fragment, which consists of the greater part of the radius, protrudes on the flexor side of the forearm, while the lower fragment, and with it the hand, assumes a position dorsal to the lower end of the upper fragment. The chief symptom can be readily deduced from this. It consists of a flattened prominence anteriorly, just above the wrist-joint, and a corresponding depression dorsally (silver-fork deformity). If the dislocation is more marked, the axis of the forearm along the outer (radial) side is angularly bent. By pressing upon the dorsal groove, the prominence grows more marked anteriorly. This is impossible with the radius intact, for the unfractured bone would resist such pressure. The position of the hand is not constant. Usually it assumes a position of ulnar flexion if the forearm is extended. Careful inspection detects some slight supination. The main symptoms, however, are sufficient: prominence on the flexor surface above the wrist-joint, a corresponding dorsal groove, and abnormal mobility. With these present, attempts to elicit crepitus torture the patient unnecessarily.

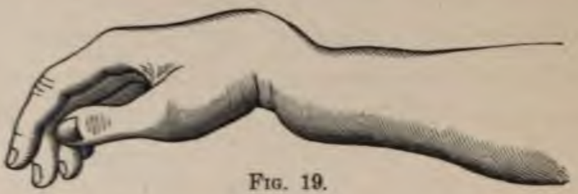

FIG. 19.

In many of these fractures the extremity shows a characteristic deformity if the flexor surface is examined. The angular break in the long axis of the limb, as indicated by the dotted line, can be seen in Fig. 20.

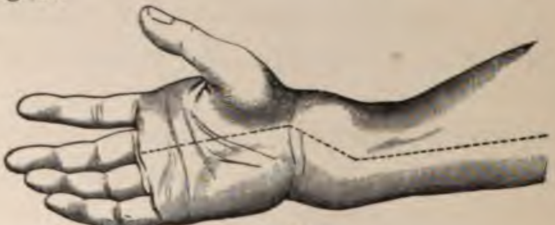

FIG. 20.

But why should this fracture be so often overlooked? When the displacement is slight the fracture is not recognised, and the condition is mistaken for a sprain; when the displacement is great, the diagnosis of dislocation is made. Surgeons in former days more frequently spoke of dorsal dislocation; in more recent days this is but rarely met with. Dupuytren was correct in stating that most of the cases of dorsal dislocation of the older authors were unrecognised fractures of the radius with much displacement.

True and undoubted DISLOCATIONS OF THE WRIST have, however, been observed. They occur between the radius and the carpus; the hand and carpus are displaced dorsally. The opposite displacement is seen much more rarely. Either of these dislocations are differentiated from a Colles's fracture by measuring the distance between the styloid process of the radius and some fixed point in the hand. In the case of the fracture, the distance remains normal; in dislocation, more or less shortening is found. Careful palpation will do the rest. In dorsal dislocation (Fig. 21) the upper convex surface of the first row of carpal bones can be felt posteriorly in the region of the wrist-joint; anteriorly the lower concave end of the articular surface of the radius can be palpated. In dislocation forward (Fig. 22) the conditions are reversed.

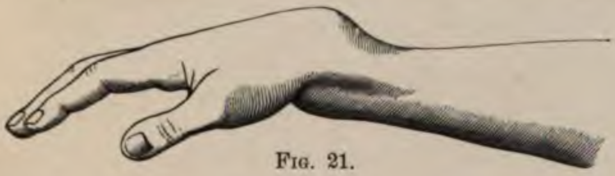

FIG. 21.

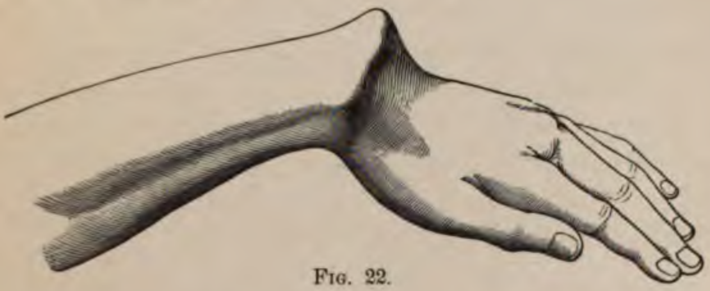

FIG. 22.

We have observed one case of *epiphyseal separation* at the lower end of the radius, in a child two years of age, due to a fall on the hand. This is a very uncommon injury.

Small children frequently sustain an obscure injury by a sudden tug upon the arm. The child keeps its arm quiet and cries aloud when the hand is grasped. The surgeon examines each joint; and, as Goyrand correctly states, finds crepitus in the elbow joint. Consequently, he prescribes cold applications to this region. As a matter of fact, the injury is situated in the wrist, and consists either of a *rupture of the dorsal ligaments*, or, as Goyrand has found, of a *dislocation of the inferior radio-ulnar articulation*. In every case extension of the hand and pressure upon the dorsum of the carpus effects a reduction, and at once relieves the pain. We need not enter into a discussion of the merits of Goyrand's view, that the trouble is due to a dislocation of the radio-ulnar joint with displacement of the triangular cartilage; but this injury must be kept in mind, as children are frequently roughly pulled by the hand. The scene described by Bogumil Goltz, of a mother, who catches her falling child with one hand and removes the boiling pot of milk from the fire with the other, is a frequent one.

In adults, *dislocation of the inferior ulnar articulation* does not often complicate the above-mentioned fracture of the radius. When it does, the attitude is characteristic. The whole hand is displaced outward and held in radial abduction; the head of the ulna is very prominent and bulging. And yet, incredible as this may sound, a man was brought to v. Dumreicher's clinic who had been treated by the doctors for articular rheumatism. The first glance showed bilateral fracture of the radii, with dislocation of the ulnæ. In addition, he had sustained a fracture of the tibia below the left knee-joint, with considerable displacement. This man had been found beneath a cave-in, and still the diagnosis of *rheumatism* had been made!

The prognosis in fracture of the radius in old people should be conservative. In very old people fissures may extend into the wrist-joint, with a resulting anchylosis. The condition may be recognised by the behaviour of the joint on the second or third day after injury. In spite of rest and bandaging, the swelling does not decrease; on the contrary, it increases, and spreads about the joint, which grows more and more painful. In a laborer I saw a T fracture of the lower end of the radius which healed without anchylosis.

Dislocation of isolated carpal bones, fractures of the metacarpal bones, and of the phalanges, require no special notice from a diagnostic standpoint. They must be recognised by palpation.

Dislocations of the thumb, however, demand special mention. Dorsal dislocation is the more frequent. The first phalanx is displaced upon the dorsum of the metacarpal bone; its axis almost forms a right angle with that of the metacarpal. The joint between the first and second phalanx is flexed. Most beginners are deceived by these symptoms. They mistake the rounded head of the metacarpal bone, which is felt bulging in the ball of the thumb, for the proximal end of the phalanx, and therefore wrongly call the dislocation anterior. By seizing the thumb and changing the position from hyperextension to a straight angle, this error can not occur, for the axis of the first phalanx will now no longer intersect the prominence, which was mistaken for the base of the phalanx. Anterior dislocation is of rarer occurrence. The first phalanx of the thumb

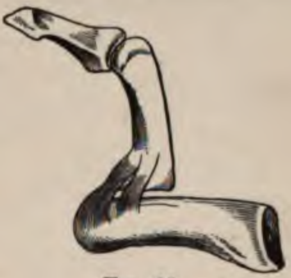

Fig. 23.

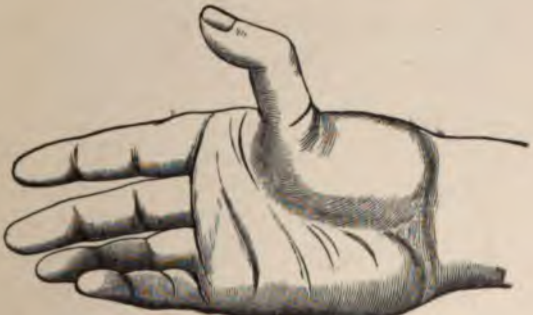

Fig. 24.

forms a prominence in the thenar eminence, and, in addition, is displaced to the radial or ulnar side of its metacarpal bone. The thumb is shortened; the interphalangeal joint is flexed. A characteristic feature is the pronation of thumb in the ulnar displacement and supination in radial displacement.

CHAPTER XIX

INFLAMMATORY PROCESSES IN THE HAND

In the vicinity of the carpus any *effusion* which is plainly recognisable can be situated only in the radio-carpal joint. The relations of such a swelling can be studied on the cadaver if the joint cavity is filled through a hole bored obliquely through the radius. Occasionally such an effusion will be seen in the living subject, and the clinical findings will then be found to correspond with those experimentally obtained. In the cases that I had occasion to see, the hand was held in slight abduction, and a flat, fluctuating swelling appeared upon the dorsum of the carpus. The upper boundary of the swellings was plainly formed by the posterior edge of the radial articular surface. The swelling showed considerable increase of tension when the hand was pressed upward toward the radius. In addition, a slight degree of abnormal mobility could be demonstrated between the radius and the carpus. In these cases, after inflammatory symptoms had subsided, absorption of the fluid took place when treatment by means of pressure was instituted. Inflammation of the carpal joints, in toto, is not distinguished by symptoms such as these, for the rigid capsules prevent the collection of a recognisable amount of fluid. Instead of a fluctuating tumour, *diffuse* swelling of the whole region occurs. The question whether the process is intra- or extra-articular then arises.

In the two varieties most frequently met with in practice, the etiology of the case will solve the problem. Rheumatism and tuberculosis are intra-articular processes; consequently, a swelling which includes the whole carpus, appearing during the course of a rheumatic attack, or evidently of tubercular origin, is intra-articular. In subcutaneous injuries there is room fo doubt, but the point at issue is of little consequence. A contusion is sure to injure the extra-articular soft parts, and may extend to the intra-articular structure as well. A sprain, on the other hand, will certainl do harm to the articulations, and may be complicate by some extra-articular hemorrhage. In these case it is of little importance to be able to state whethe the one or the other of the many complicated structures which form the hand have been harmed, outsid or inside the articular cavities.

In wounds and cellulitis this statement does no hold true. It is necessary to decide whether the woun has penetrated the joint, whether the pus has erode the capsule. The probe at once answers these questions. In cases of longer standing, in purulent processes due either to open wounds or to cellulitis, abnormal mobility of the carpus and crepitus will sho whether the ligaments have been destroyed and th articular cartilages eroded.

Tuberculosis of the Carpus, which was previousl merely alluded to, gives an unmistakable picture. Th hand assumes a position of slight flexion, and is supported by the patient. All the phalangeal joints ar fully extended; this is due to prolonged disuse. Th carpus is greatly swollen; at times it is wider than the metacarpus. Œdema covers the dorsum of the carpus and extends upward for some distance upon the forearm. The hand is like that of a giant, and the picture is completed by numerous granulating fistulæ situated upon the back, outer, and inner side, which discharge thin pus. Later, mobility and crepitus can be obtained in many instances.

Earlier stages of tuberculosis of the carpus can be recognised by the following symptoms: the appearance of the patient, spontaneous origin of the trouble, slow course, and diffuse swelling devoid of the cardinal signs of inflammation. The original focus of disease is frequently situated in the lower end of the radius or in the base of a metacarpal bone. Therefore, at this early stage, in addition to diffuse swelling of the carpus, the disease is more evident at the lower end of the radius or the base of some metacarpal, as the case may be.

In acute processes, at the lower end of the radius, the diagnosis between rheumatism and osteomyelitis may be made by means of the symptoms already mentioned in connection with these same diseases in the elbow-joint. It should be kept in mind that the radius is a more common site of osteomyelitis than the lower end of the humerus.

SYNOVITIS of the tendon sheaths of the wrist is a special variety of periarticular process. Anteriorly a large synovial sheath is common to the flexors of the fingers, and external to this is the separate sheath of the flexor longus pollicis. The sheath extends about two centimetres up the forearm and passes beneath the anterior annular ligament. Its lateral boundary may be indicated by a line extending diagonally from the base of the thenar eminence to the metacarpophalangeal joint of the little finger. A swelling of this tendon sheath must consequently possess the following characteristics: 1, boundaries corresponding to the above description; 2, a narrower middle portion, where the annular ligament constricts the tumour, so that the portions in the forearm and in the palm are wider. If the swelling fluctuates, we are dealing with *tenosynovitis*, and as a rule many small ricelike bodies can be felt. If the tumour is merely elastic, the condition is one of so-called *fungus* (tubercular tenosynovitis)—that is, proliferation of granulation tissue without suppuration, formerly merely surmised, but now positively known to be of tubercular origin. Suppuration of the tendon sheath is accompanied by high fever, bluish-red discoloration of the skin of the palm, and œdema of the dorsum of the hand. The pus, by preference, burrows along the tendons and spreads upward upon the forearm. Occasionally a panaritium will extend in a similar fashion, and may therefore be discussed in this connection.

The word PANARITIUM (whitlow) originally included only an inflammation of the nail proper, but the term has gradually been applied to all suppurative inflammations of the fingers. Not including the inflammations of the nail bed, which are characterized by strict limitation to the structures of the nail, three forms of inflammation are met with. They are *phlegmon of the cellular tissues*, *suppuration of a tendon sheath*, and *periostitis of a phalanx*. The following points are of value in their diagnosis: *Periostitis*, as a rule, will affect an entire phalanx, causing a uniform enlargement of its whole circumference. *Suppuration of a tendon sheath* will correspond to the anatomical distribution of these structures, and consequently appear only on the flexor surface and extend along all the phalanges. A *cellular phlegmon* will not remain confined either to a single phalanx or to the tendon sheath; its distribution will correspond to the cellular tissues irrespective of anatomical boundaries. In a given case, a glance will suffice to arrive at the correct diagnosis. If the swelling and redness extends along the radial side of a phalanx, or along the dorsum of two phalanges, or, in another case, along the flexor and ulnar surface of two phalanges, we may suspect that the inflammation affects the cellular structures, and not some anatomical unit. The distribution of the periosteum and cellular tissue requires no description, but the anatomy of the tendon sheaths deserves a few words of explanation. The sheaths of the flexor tendons are divided into two compartments—the phalangeal and the carpal. The phalangeal tendon sheath is a cylindrical tube, through which the tendon passes like a trochar through its cannula. It ends blindly above, and is attached to the tendon. Movement is permitted by reduplications of the membrane, which folds and unfolds in response to motion. The upper limit of the synovial sheath is slightly distal to the middle of the palm. A suppuration of the sheath, consequently, extends from the terminal phalanx, along the flexor surface of all the phalanges, to the middle of the palm.

The thumb and little finger do not conform to this arrangement. The sheath of the little finger is not shut off above; it is directly continuous with the large carpal tendon sheath. The thumb boasts of a privileged and independent position, compared with that of the other fingers, for its carpal sheath is distinct and continuous with that of its phalanges. For this reason, inflammation of the tendon sheaths of the little finger and of the thumb are more serious, as the process can readily extend to the great carpal sheath.

When the suppuration extends from the tendon sheath of the little finger along the small opening which is situated between the superficial and deep flexor tendon, and spreads to the large carpal sheath, the involvement causes an increase in the severity of the symptoms. The inflammation rapidly involves the whole palm, and spreads up to the lower part of the flexor surface of the forearm, passing under the annular ligament. The palm becomes a dark coppery red, and an extensive collateral œdema of the dorsum of the hand at once develops.

Suppuration of the other phalangeal sheaths may, however, also reach the great carpal sheath, not by direct extension, but by dissecting its way along the tendons themselves. The pus does not break through the dense palmar fascia, but points at the dorsum, after burrowing between the metacarpal bones. Most commonly it finds its way to the interval between fourth and fifth metacarpal, close to their heads; but at times, as has been stated, it may appear on the forearm.

The diagnosis of these various conditions can usually be made. If asked for a prognosis, be cautious, and do not limit the extent of the sloughing to a tendon sheath, if the process was originally a suppurative tenosynovitis. For it is not possible to predict whether or not a small piece of bone, or even an entire phalanx, may necrose.

At times it is impossible to decide whether a suppurative tenosynovitis or a periostitis is, or has been, in progress. After the first symptoms have subsided, an enlarged, sausage-shaped finger, with two or three fistulous tracts opening on the palmar surface, results. Usually one or all the phalanges are found necrotic in these cases. But, exceptionally, suppuration of the tendon sheath, accompanied by enormous thickening of the finger and numerous fistulæ, may run its course without injury to the bones. It is, therefore, necessary to probe the sinuses, and test for abnormal mobility of the phalangeal joints, before giving a prognosis. If one or all the joints show abnormal mobility, with faint crepitus, and the sinuses do not lead to roughened bone, anchylosis, without separation of a part of the phalanx, may be the final outcome.

The common carpal extensor sheath is infrequently the seat of suppurative processes. It is more often distended by a non-purulent effusion. The sheath is small, and ends toward the metacarpus in four small processes. The shape of the resulting swelling is quite characteristic.

Serous effusions into the various tendon and synovial sheaths of the carpus are readily recognised, if their distribution and extent are kept in mind. As they are unimportant, a more detailed description may be dispensed with. But one variety deserves mention. A patient complains of pain in his hand while engaged in his work. He is unable to locate the painful spot precisely. Examination of the wrist-joint shows no swelling, no inflammation, and no change in the soft parts. The case may be ascribed to rheumatism or even to malingering. Another physician may, purely by chance, take hold of the forearm while examining the mobility of the wrist-joint, and thus discover the seat of the trouble; for the examining hand, which steadies the forearm, feels a peculiar crepitus. The disease is an inflammation of the tendon sheaths, accompanied by the development of *ricelike bodies.* They most commonly are found in the sheaths of the three muscles of the thumb which run obliquely across the lower part of the extensor surface of the forearm.

In conclusion, we may discuss a variety of small swellings found on the hand—GANGLION. Until recently three varieties were distinguished. On the cadaver of adults, small colloid cysts, adherent to the joint capsules, are found. Their size rarely exceeds that of a nut. They are called *cyst ganglia.* We further observe preformed diverticula derived from the joint capsule, or pathological diverticula, which are distended with viscid fluid and form the joint ganglia. Finally, a tendon sheath may be distended (hygroma), or, if only a part of the sheath or some diverticulum is dilated with fluid, a ganglion of the tendon sheath results. This was the accepted teaching until recently.. Therefore, it was customary, first, to decide the variety to which a ganglion belonged. If a ganglion is situated between the tendons, can not be emptied and does not change in tension when manipulated, it is a cyst ganglion. If, however, it can be emptied by pressure, or at least grows less tense upon manipulation, it is a joint ganglion. About the wrist-joint ganglia usually are of the cystic variety. These small cysts, according to Payr, are the result of small inflammatory foci. Ganglia arising from the tendon sheaths do not exist.

In rare instances a hygroma may be of acute origin, most probably due to a preceding dry tenosynovitis (with rice bodies) of the corresponding tendon sheaths. To open such a swelling, under the impression that it is an abscess, would constitute a grave error. Keep in mind that in acute hygroma, though accompanied by much pain, the swelling is not great, the skin not reddened, and the pain not of the intense throbbing character met with in abscesses.

Tuberculosis of the dorsal sheaths follows the same course as that already described in connection with the great carpal sheath. We have likewise observed " fungus " of the phalangeal tendon sheaths. When rupture occurs, it is accompanied by pain, but no pus is discharged. The fistula may heal, but the elastic swelling persists. New sinuses may develop later.

CHAPTER XX

A FEW REMARKS ON THE DIAGNOSIS OF ABDOMINAL TUMOURS—ABSCESSES OF THE ABDOMINAL REGION

WHAT help will the topographical situation of an abdominal mass give us, if all other factors are left out of account? The tumour may be preperitoneal, intraperitoneal, or retroperitoneal.

In general, the following remarks will hold good. A retroperitoneal mass is not movable. An intraperitoneal growth is movable, and its motion is influenced by respiration in a manner similar to that of the intraperitoneal organs. A preperitoneal tumour is movable, but does not move up and down with respiration like the diaphragm, liver, spleen, etc., but its arc is forward and backward like that of the abdominal wall.

Any one familiar with abdominal palpation is well aware that it may frequently be difficult to decide in what direction a mass is moving. Other means of arriving at a conclusion must, therefore, be applied.

If a tumour is preperitoneal, it must lie in the subserous layer, in front of the transversalis fascia, between or in front of the muscles. In any of these situations, however, the relation of the tumour to that part of the abdominal wall to which it is attached must remain the same, no matter what position is assumed by the patient. If the abdominal wall is so lax that the tumour moves with change of position, the abdominal muscles must be made tense in order to prevent shifting. If this manœuvre enables us to determine that the tumour is movable, it is undoubtedly intraperitoneal. Such a change of position (when the abdominal muscles are held tense) can not be detected by palpation, because the contracted muscles obscure the outline to the palpating fingers; but percussion enables us to note change of position. For instance, a long-pedicled cyst of the left ovary may gravitate toward the right. If the patient is placed upon her right side and ordered to contract her abdominal muscles, it is then found that the dulness has shifted from left to right.

A tumour situated behind the layer of muscles disappears, or is at least flattened, when these muscles are contracted; and, furthermore, the tensely contracted muscle layer can be felt passing over the mass. A tumour placed in front of the muscles is pushed forward and grows more distinct under these conditions. If the tumour is freely movable when the abdominal wall is rigid, its attachment is anterior to the fascial coverings of the muscles—or, in other words, subcutaneous.

So far the discussion has dealt purely with diagnosis in the abstract. In practice, other factors must be considered from the very outset of the examination.

In the first place, the situation and outline of the tumour may be such as to permit of only *one* interpretation. A tumour of the left hypochondrium, which juts out from beneath the ribs, with its greatest convexity posteriorly, allowing a notched edge to be felt, will at once impress us as an enlarged spleen.

Secondly, the size and most striking physical characsiderations will govern our reasoning. A pyloric carcinoma will never reach the size of a head; a bony, hard tumour of the uterus will prove a calcified myoma.

Thirdly, especially when dealing with a smaller tumour, its relation to certain organs may be so apparent that the possibilities to be considered are at once narrowed to certain limits. If we find a small, hard mass in the region of the pylorus, accompanied by dilatation of the stomach, we at once suspect a pyloric cancer.

The functional disturbances which accompany various tumours and occupy the foreground of the picture must also be considered—vomiting in pyloric carcinoma, intestinal obstruction in cancer of the gut, the metrorrhagia and menorrhagia met with in fibroids of the uterus.

In addition, lesions situated at a distance must be taken into account: for instance, a kyphosis of the lumbar spine in a patient with a fluctuating tumour of the lower part of the abdomen. We must at once decide whether we are dealing with a cold abscess which took its origin from the diseased vertebræ.

Finally, the history may show whether the mass is inflammatory or neoplastic. A tumour which appears during the puerperium, accompanied by fever and pain, will at once impress us as an abscess originating from the genitals or their vicinity. A tumour which develops slowly during the course of years, without known cause, unaccompanied by fever or pain, will prove tó be a neoplasm.

To sum up, the differential diagnosis will usually narrow down to two or three possibilities.

Abdominal ABSCESSES will be considered first.

In addition to the previously mentioned topographical signs which determine in what layer the abscess is situated, certain typical modes of extension will go far toward proving the origin and nature of such a swelling. After Henke had enriched our knowledge of the exact anatomy of the cellular planes, other surgeons—König, Soltmann, Schlesinger—for experimental purposes, injected substances into the tissue planes at sites where certain typical abscess formations were most common. The results of these experiments corresponded quite closely with the clinical picture obtained in the sick-room, so that we may state that certain abscesses spread in directions which may usually be anticipated.

Further assistance is furnished by the initial symptoms, the course, and the relations to neighbouring organs.

For example, let us consider a patient who complains of fever, constipation, and vomiting, followed by the sudden appearance of a tumour of the *right* iliac fossa which is exquisitely tender to pressure. Inspection shows little or no bulging of the affected region; the skin is entirely normal. Percussion demonstrates tympany over the mass; more careful palpation shows the mass to be immovable and fixed. We may then conclude that the tumour is retroperitoneal, for the cæcum is placed anterior to it. When dealing with other retroperitoneal tumours, it is found that the intestine is pushed aside, and the tumour projects forward to the abdominal wall. The cæcum, however, can not be forced to the side, and consequently a tympanitic note is heard. Other symptoms may aid us. The patient may feel pain radiating downward to the thigh, and the thigh may assume a position of slight flexion. Or, if pressure upon the iliac vein causes œdema of the leg, it is another confirmatory sign of the retroperitoneal seat of the affection. This variety of inflammation was called PARATYPHLITIS by Oppolzer. The abscess may burrow upward to the kidney, downward to Poupart's, or even beneath the ligament, by following along the blood-vessels.

Very similar symptoms may be due to a circumscribed peritonitis confined to the neighbourhood of the cæcum, except that its onset is stormier and more violent. Oppolzer designates it PERITYPHLITIS. He emphasizes the fact that the percussion note is *not* tympanitic, and that the tumour may be pushed downward slightly, for it rests upon the anterior surface of the cæcum, supported as if upon an elastic cushion.

When the mass can be palpated, as described above, it is evident that the diagnosis between perityphlitis and paratyphlitis is possible. As a rule, the excessive tenderness of the whole region will prevent such extensive palpation, and the course alone will show whether we are dealing with a circumscribed abscess of the retroperitoneal tissues (retrocolic) or with a circumscribed peritonitis developing eventually into a general perforative peritonitis.

Since, in the last few years, surgeons have operated more and more frequently in these two varieties of inflammations, they have assumed a constantly growing importance. As their special pathology has been more carefully studied, it has been proven that both paratyphlitis and perityphlitis are nothing more than the two chief outcomes of inflammation and ulceration of the vermiform appendix. A generic name was sought for—Nothnagel proposed *Skolikœiditis*; Eiselt, the appellation *Epidesmitis*; in America, the name *Appendicitis* is uniformly employed.

Let us discuss another case. During the puerperium the patient has a chill (this may be wanting); the temperature rises, and spontaneous abdominal pain also appears; in addition, the lower part of the abdomen grows so acutely tender that a bimanual examination can not be made. Meteorism and vomiting occur in the further course of the trouble. Here no doubt can be entertained: it is evidently a case of perimetritis or PARAMETRITIS. If the patient does not die, we can decide later whether the abscess which forms is intraperitoneal or extraperitoneal. If the tumour, by vaginal examination, proves to be situated to the side of the uterus, but closely connected with it, or separated only by a shallow groove, it is, as a rule, extraperitoneal; likewise if the mass extends into the iliac fossa. In many other cases the question can not be satisfactorily answered.

In a great number of cases we discover an abscess of whose existence both patient and relatives are entirely ignorant. This, of course, applies to the slowly developing gravitation abscesses seen in Pott's disease of the spine. The children are brought to the clinic by their parents to be examined for weakness of the lower extremities or for a developing gibbus. These cases are of daily occurrence, and the iliac fossæ are examined quite as a matter of course, to see whether a PSOAS ABSCESS has developed. If the caries of the spinal column has been recognised, the psoas abscess can be diagnosed by the following points: 1, considerable flexion of the thigh upon the pelvis; 2, free mobility of the hip-joint except to motions which extend the thigh upon the pelvis; 3, an increased sense of resistance to deep palpation above Poupart's ligament with an elongated tumour along the psoas muscle. In some cases the symptoms are even more striking. We then find dulness to percussion and fluctuation above Poupart's, œdema of the skin beneath the ligament, and not infrequently a fluctuating swelling, from which fluctuation can be transmitted to the abdominal mass. Psoas abscesses may point laterally along the crest of the ilium; this is much less common. Rarely they burrow in the subserous tissues, or gravitate downward to the glutei and other muscles of the buttock. In all these forms the psoas contracture is the one symptom to be relied upon.

Of special interest are the SUBSEROUS ABSCESSES of the iliac fossa. They lie between the peritoneum and the iliac fascia, and develop either stormily, or quietly without pain or febrile symptoms. As a rule, these abscesses are due to tuberculosis of the spine. They vary largely in outline and size, both these factors depending on the site of the abscess. In some cases they appear along the anterior abdominal wall, others develop along the posterior wall, and still others along the true pelvis, so that they may be palpated per vaginam or rectum. The subserous location of the abscess may be recognised as follows: the deeper location is excluded by absence of psoas contracture and by the more plainly marked bulging of the mass. Intraperitoneal abscesses cause peritoneal symptoms during their development, and are not so entirely fixed as to resist palpation or the respiratory movements of the viscera. The subserous location of the abscess can be positively demonstrated when it develops along the anterior abdominal wall. The parietal peritoneum is then slowly pushed upward by the growing abscess; with it the respiratory line—i. e., the line which marks the limit of the respiratory movement of the abdominal wall—is displaced upward. Below this line we find dulness and fluctuation. Here the abscess may be boldly opened. On account of the danger of secondary hemorrhage, it is usually preferable to invade the deeper-lying parts from the outset.

In some cases psoas abscess may occur without accompanying psoas contracture. Its elongated form will serve to distinguish it from subserous abscesses.

Of great importance are the so-called SUBPHRENIC ABSCESSES (Leyden), especially since Maydl has given a careful exposition of their course. These abscesses may be due to various causes; Maydl describes no less than twelve groups. They arise most commonly from the alimentary tract, from the appendix, or from the kidneys. The diagnosis must, therefore, be based upon some antecedent primary abdominal trouble (or injury), which is followed by the symptoms of a deep-seated purulent focus (continuous fever, collateral œdema on the affected side, elastic resistance of a deep swelling). In the second place, symptoms referable to an involvement of the diaphragm should be looked for (hiccough, pains radiating to the shoulder). Physical examination shows dulness or flatness of the lower part of the thorax on the affected side. The line of flatness rises and falls with respiration—in pleural effusions the line is fixed.

The experience of the last few years has shown that an abdominal abscess, which runs an atypical course, must cause us to think of ACTINOMYCOSIS, for this disease appears in various ways. For instance, an actinomycotic focus in the anterior abdominal wall between umbilicus and pubes. Spontaneous appearance, chronic course, marked induration, with stony, hard periphery, a central fluctuating area—these are the symptoms of a typical case. The induration, which resembles that of a phlegmonous process, is not in keeping with the painless fluctuation. The central painless fluctuating area reminds us of a cold abscess, but the infiltration of the edges does not harmonize with this diagnosis. In other words, a *combination of the symptoms of acute and chronic inflammation.* In those cases in which the process involves the posterior abdominal wall, multiple deep-seated foci are present. We here find many fair-sized masses, hard, with occasional softened areas, but tuberculous signs are absent in other parts of the body, and no peritonitic history can be obtained. Chronic abscesses of the navel should be regarded with suspicion; they frequently prove to be of actinomycotic origin.

CHAPTER XXI

A FEW SURGICALLY IMPORTANT ABDOMINAL TUMOURS

A WOMAN presents herself for examination on account of a large abdominal tumour. The tumour is of such size that the patient resembles a woman in the last months of pregnancy. The exposed abdomen is seen to be uniformly rounded, the navel not protruding, and the abdominal walls in no way abnormal. Inspection shows that we are not dealing with ascites, for in ascites the abdomen is not round—it is broadened; the fluid gravitates into the flanks when the patient lies down, and the intestines are floated upon the surface. This causes the flanks to broaden and the umbilicus to protrude.

The enlargement of the abdomen might be due to distention of the intestine by gas—intestinal meteorism. In this condition, if the abdominal wall is lax and the subcutaneous layer thin, intestinal coils may be distinguished. If, on the other hand, the walls are tense and the fatty layer well developed, the abdomen has a full, rounded appearance. Percussion will help us out, for if the note is tympanitic over the entire abdomen the distention is due to meteorism, whereas if the note is flat over a considerable area of the abdomen we are dealing with a tumour.

In hysterical women *Phantom tumours* are met with; a rounded protrusion of the lower part of the abdomen is thus designated. This gives the patient the appearance of pregnancy. Palpation over the site imparts a marked feeling of resistance, which may lead to the belief that the case is one of pregnancy or tumour. But the percussion note is tympanitic, and, under anæsthesia, the tension and protrusion disappear. The whole is a phantom—hence the name.

A flat note merely indicates absence of air, but does not determine whether it is caused by a solid or fluid. If fluctuation is obtained over the entire area of flatness, we are, of course, dealing with fluid.

We next determine the limit between flatness and tympany, and mark the line along which the change in note is found. The flat area has a convex margin above and also toward the right side; but one flank remains tympanitic. *Ascites* does not give such a boundary-line, for the change in note caused by free fluid is along a line with its concavity directed upward, and *both* flanks are flat. The convex outline of the flat area (convex above and limited to one side) leads us to suspect encapsulated fluid, contained, most probably, in a sac. In order to prove this, the patient is turned upon her right side. We now find that the right flank, which was

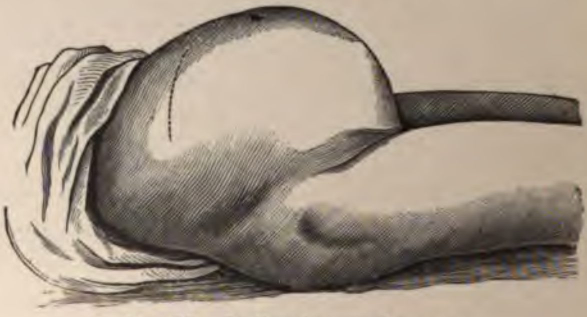

FIG. 25.—Unilocular ovarian cyst.

tympanitic in the dorsal position, continues to remain so. If the fluid were ascitic, it would gravitate into the right flank, with the result that the left side would become tympanitic and the right grow flat. In our patient the note remains unchanged in spite of changes of position. This definitely proves that the fluid is not in the free peritoneal cavity, but inclosed in a limiting membrane.

Percussion has further shown that a tympanitic area separates liver and spleen not only from the tumour in question, but also from each other; consequently the mass can have no connection with these organs. The outlines of the stomach, also demarcated by percussion, show no connection between stomach and tumour. The three mentioned organs were found somewhat higher up than normal, the diaphragm and intestines also being pushed upward by the growth which occupies the lower part of the abdomen. As the flatness has no determinable lower limit, it probably extends into the pelvis. If the area of flatness had been centrally located, with a convex margin toward the left as well as toward the right flank, and tympany in both flanks, two

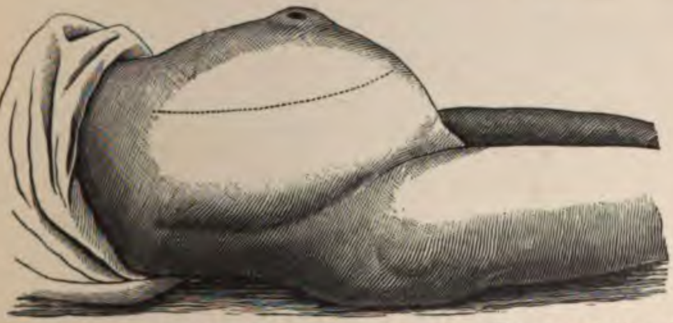

FIG. 26.—Abdomen in ascites, seen from the side. (The dotted line shows the upper limit of the fluid.)

other conditions would require to be considered—*pregnancy* and *distended bladder*. These two conditions must be kept in mind, because it has happened that pregnant women have had laparotomies for abdominal tumour performed upon them when nothing but a pregnant uterus was present, and that women who merely had retention of urine were placed upon the operating-table by surgeons labouring under the same mistake. Let us, therefore, never forget that these two conditions would, if overlooked, cause grave risks—risks chiefly to the surgeon's reputation. But in our case the flatness extends into the left flank, and tympany can not be elicited, even in the loin.

As ovarian cysts are of common occurrence in women, it may be suspected that the mass is a CYST of the left OVARY. By vaginal examination, the uterus is found movable, normal in situation. The menses are scanty, but regular. The tumour has developed in the course of a single year. The patient noticed, at the outset, that the lower part of her abdomen increased in size on one side only. No pain was present at any time. Examination per rectum, by Simon's method (under anæsthesia), if made, would show the right ovary present, the left out of reach.

This was a typical instance, but in practice we do not always meet with clear cases, obscure ones being more often encountered.

Let us now turn to a case in which the abdomen is distended by an enormous quantity of ascitic fluid. Here it may occur that the fluid lies in front of the intestine, because the intestine is held down by the mesentery, and can not reach the anterior wall of the abdomen. This would give flatness anteriorly. The same might happen in chronic peritonitis, where the intestine and mesentery are bound down to the posterior wall by adhesions. TUBERCULOSIS of the PERITONEUM presents such a picture. Serous fluid accumulates in the peritoneal cavity, frequently in large quantities. The intestines are matted together, and single coils are often adherent to the posterior abdominal wall. As the fluid is situated in the relatively free peritoneal cavity, change of note occurs on change of position; but the phenomenon takes place more sluggishly, and not as completely as in ascites. Within the flat area, isolated islets of tympany remain, and may be found to vary in clearness and extent at different examinations (distention of adherent intestinal coils). Further evidence is furnished by the emaciation, habitus, hereditary taint, and occasional fever. A primary cause for ascites is also lacking. Occurring in women, with the fluid limited to the lower and central portion of the abdomen, and the adherent intestine laterally situated, the picture of an ovarian cyst may be simulated. But in such cases the tense feeling encountered in cysts is wanting, and the impression produced by a distinct limiting membrane is absent. Both ovaries may perhaps be felt; some change of note can be obtained; fever and emaciation are present.

Even in ovarian cysts, the note anteriorly may be tympanitic if a communication exists between the cyst and a loop of intestine.

The history of the case must be considered. Aspiration may help in the diagnosis, if the contents is examined microscopically and chemically. Paralbumin speaks for ovarian cyst; fibrin and lymphocytes for ascites; epithelial cells for a neoplasm.

Encapsulated intraperitoneal fluid may very closely imitate the picture of an ovarian cyst. The most expert ovariotomists have been deceived, and found encapsulated fluid, cancer, or tuberculosis of the peritoneum upon opening the abdomen. Spencer Wells calls attention to the fact "that all cases in which the abdominal wall is thin, tense, or œdematous; in which anasarca, general emaciation, cachexia, and free fluid in the peritoneal cavity is present, should awaken suspicion, especially if the emaciation and pain are marked and show a rapid increase."

Of equal importance is the differential diagnosis of ovarian from RENAL TUMOURS. In some cases the diagnosis is not possible. From my student days I remember a case, which the most renowned clinical teachers in Vienna mistook for an ovarian cyst, and which, at the post-mortem, proved to be an enormous hydronephrosis. As a rule, the diagnosis can be definitely made; quite frequently it is easy. The following must be kept in mind: As the renal tumour is retroperitoneal, and starts from the region of the kidney, it extends from behind and above, downward and toward the median line, crowding the intestine before it. An ovarian tumour forces the intestine behind it. The hydronephrosis has the colon passing over its surface; this can be demonstrated by filling the gut with air. The flanks always percuss flat, and fluctuation is obtained in the distended loin as well as in the flank and anteriorly. A hydronephrosis can empty itself suddenly or periodically by way of the bladder. This may also happen with ovarian cysts if they rupture into the bladder and empty their contents into this viscus. Examination of the fluid will clear up the diagnosis in these cases. Renal tumours are accompanied by disturbances of the kidney function, such as hematuria, albuminuria, renal colic, etc.

Recently, movable hydronephrosis and movable renal tumours have been reported. To understand these, it is only necessary to imagine the diseases in question occurring in a movable kidney.

A solid renal tumour may be recognised by the following characteristics: It is situated in the lumbar region, protruding well into the flank. Anteriorly it is overlapped by the colon, which may be recognised by its tympanitic note, and the gurgling obtained on palpation. Only in rare instances does the mass extend to the median line, and in no case does it move with respiration. With one hand placed anteriorly on the abdo-

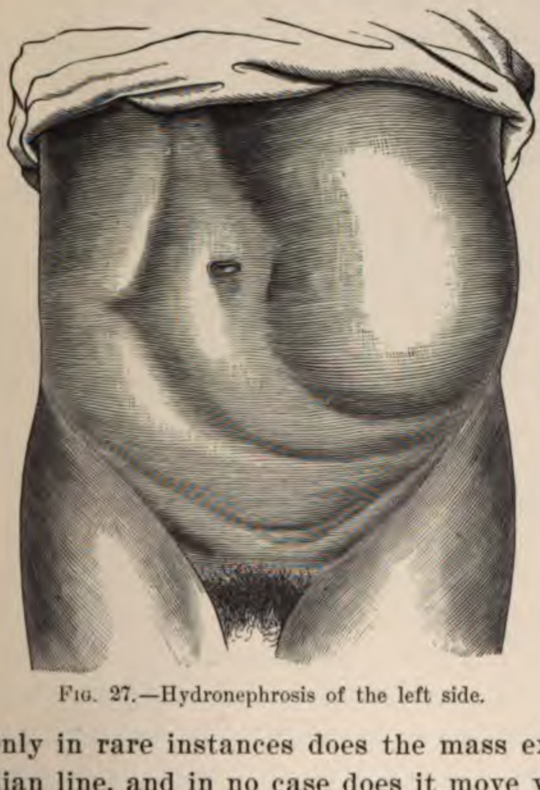

FIG. 27.—Hydronephrosis of the left side.

men and the other posteriorly in the flank, pressure from before backward is communicated to the posterior hand, and causes the flank to bulge (*ballottement*). If the tumour is on the left side, we must take into account that it may arise either from the kidney or from the spleen. But splenic tumours rise and fall with the respiratory movements, always retain the shape of the spleen, and *have an area of tympany behind them in the loin.* As a rule, their anterior margin is sharper. Both varieties of tumour are situated above the pelvic brim.

In another case, inspection shows that the tumour is knobby and uneven, and palpation proves it to be hard and solid. This nodular tumour, without a shadow of doubt, extends into the pelvis, and, from the patient's story, has advanced from below. Two varieties of new growths must be considered—either a uterine myoma or a multilocular ovarian cyst, in which the connective tissue has developed at the expense of the fluid elements. A UTERINE FIBROID is recognised by its extreme hardness, which in spots is almost bony. Even more characteristic is the marked, often enormous, elongation of the uterine canal, readily demon-

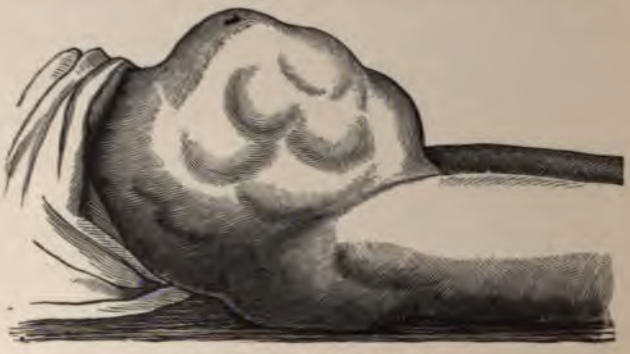

FIG. 28.—Multilocular cyst.

strated by the sound. This elongation is caused by the growth of the tumour, which stretches the uterus longitudinally. The portio vaginalis, examined bimanually, accurately follows all the movements executed by the abdominal mass. The development of the fibroid is accompanied by dysmenorrhœa, metrorrhagia, and menorrhagia.

When dealing with tumours of the lower part of the abdomen—I want to emphasize this point once more—never forget to think of the bladder. If the patient is a woman, past puberty, think of pregnancy. Spaeth was wont to say that the bladder was the most dangerous organ encountered by the gynecologist—dangerous both to his reputation as a diagnostician and operator. Ovarian cyst has been diagnosed, and pregnancy found; an overdistended bladder has been mistaken for uterine fibroids; and such mistakes have happened to the most experienced. And have not fæcal masses been taken for solid tumours, strange as this may appear to the beginner? To avoid these mistakes, think of the bladder, think of pregnancy, remember fæcal masses—nothing is more readily forgotten.

In the upper segment of the abdomen, echinococcus cysts of the liver or spleen, and pancreatic cysts, are the tumours most readily confused with tumours arising from the pelvic organs. If this appear incredible, remember that echinococcus cysts of the liver may occupy almost the entire false pelvis, and, on the other hand, ovarian cysts, with long pedicles, reach to the lower surface of the liver.

Echinococcus of the liver frequently appears as a flattened, bulging tumour, which is elastic or fluctuating, and projects from the anterior surface of the liver. In such cases it is unmistakably attached to the liver, and can be confused with scarcely any other condition, unless we consider abscess of the liver. Such abscesses, in addition to their rare occurrence, are accompanied by more threatening symptoms. If the cyst is larger, its fluctuation is readily demonstrated, and its intimate connection with the liver, in spite of change in position, is readily recognised. The dulness produced by the tumour passes uninterruptedly into liver dulness, following the liver in respiration. The pelvis is not occupied by the mass. Echinococcus of the spleen increases the area of splenic dulness. The enlargement, however, does not retain the typical shape of the spleen, characteristic of other tumours of that organ, which enlarge downward and inward. The dull area, for instance, is broad, and extends both in front and behind; fluctuation can be obtained.

Pancreatic cysts are usually placed transversely across the upper part of the abdomen. The stomach, recognised by its tympany, is anterior to the cyst, the transverse colon below it. If the colon is distended per rectum, either with air or water, the gut will be found coursing along the lower margin of the pancreatic cyst, like a festoon.

Of interest are the *tumours of the abdominal wall*, especially the so-called Nélaton's tumours; sarcoma or fibroma, which spread either from the anterior superior spine of the ilium, or from the neighbourhood of the costal margin, and may reach a considerable size. They follow the anterior abdominal wall in respiration—i. e., from before backward (dorsoventrally)—and not the diaphragm, which moves from above downward. If not of large size, it is possible to grasp their base and notice that they become fixed when the abdominal muscles are contracted. These tumours should always be kept in mind, for, especially if small, they may simulate graver conditions, such as a pyloric tumour or tumour of the gall-bladder.

The rules given in this chapter will be found to apply to typical cases. We may expect, however, that a further increase in our experience will make diagnosis easier.

CHAPTER XXII

HERNIA

THE conception of hernia demands (1) a hernial contents, (2) a hernial sac, (3) a hernial orifice; these three requisites should, therefore, be demonstrated to make a diagnosis theoretically complete. In simple cases all three may be readily recognised. Assuming that a tumour is tympanitic, increases in size under the influence of intra-abdominal pressure (coughing, straining, etc.), and is reducible, we are warranted in diagnosing that the mass contains not only a viscus, but a definite viscus —the intestine. After reduction, a flabby sac is left behind, which glides smoothly to and fro under our fingers, and thus indicates the presence of two serous surfaces in contact. This is the hernial sac. Finally, on inverting the skin, we may pass our finger along the canal, until the tip reaches the abdominal cavity (coughing imparts the impulse of the intestine to the finger). The configuration of the hernial canal and hernial orifice can now be examined. In such a case no difficulty arises in the diagnosis of the condition. If we assume that the tumour is not reducible, it at once becomes impossible to demonstrate the hernial sac. If, in addition, the tumour is not tympanitic, as would be the case if the contents is omentum or ovary instead of intestine, we can no longer positively determine whether the contents of the tumour is one of the many intra-abdominal viscera. The tumour may be so broad at its point of attachment that its base can not be reached; therefore definite proof of the existence of a hernial orifice is wanting. Although in many, and in fact in most, cases it is easy to demonstrate the existence of a hernia, instances do occur in which extensive examination is required. If, for example, the tumour under discussion can not be shown to be one of the abdominal viscera, another point of evidence must weigh heavily in the scale—such as its extension into a hernial orifice, and eventually into the abdominal cavity. Vice versa, if it can be shown that the tumour contains some abdominal viscus, this evidence is sufficient, even if the hernial opening or sac can not be found. It is clear that the last instance is more directly convincing. The method of recognising abdominal viscera in a hernia will, therefore, be detailed.

The ordinary hernia, either small or medium-sized inguinal, femoral, or umbilical, may contain intestine or omentum, or both. *Omentum* gives the impression of a mass composed of strands, with small nodules scattered along them. This is most plainly felt in umbilical herniæ. Reduction is gradually accomplished by replacing small portions at a time. The percussion note is dull. If a loop of gut has prolapsed into a hernia, the note is tympanitic over the whole or part of the tumour. The patient notices gurgling sounds in the hernia, and reduction occurs rapidly, accompanied by the same gurgling. In entero-epiplocele these symptoms are found combined, for the omentum either lies in front of the intestine, as it does within the abdominal cavity, or envelops the gut. An inguinal hernia, or, less frequently, a femoral, may contain the *ovary*, the outline of the body felt then resembling the form of this organ. Extensive movements of the uterus are accompanied by movements of the hernial contents. In addition, it has been observed that the prolapsed ovary is tender to pressure, and in some cases enlarges and grows painful at the time of menstruation.

Hernia of the bladder may occur in one of two ways. Either the fundus prolapses into a true hernial sac, or a lateral, non-peritoneal portion of the bladder forces its way along the subserous layers. In either case, compression of the tumour causes strangury, the size of the tumour diminishes on urination, and a catheter introduced into the bladder may be guided into the hernial swelling.

More rarely the *stomach* enters into the formation of a hernia, always in company with the omentum and transverse colon. The diagnosis can be made if part of the tumour enlarges immediately after taking food, and then grows flat to percussion. Swallowing of liquids should be accompanied by gurgling sounds.

Large eventrations may contain several abdominal organs. In fact, a whole human being, or even two, may be contained within the hernia. The uterus may lie in the hernia, become pregnant, and remain there during the whole period of gestation. No such cases have come under my direct observation. In other cases part of the liver, the stomach, etc., may form the contents. It is not difficult to reason out methods of recognising these various viscera, for the contained viscus is no longer found at its normal site. The area usually occupied by the suspected viscus must be examined by palpation, percussion, etc., and its absence demonstrated.

In discussing the more common varieties of hernia, it becomes necessary to consider with what other conditions, inguinal, femoral, and umbilical herniæ may be confused.

INGUINAL HERNIA, from the point of view of diagnosis, may be divided into two chapters. Incomplete herniæ—i. e., such as are still within the inguinal canal, or in its immediate vicinity—may be confounded with a small group of tumours, namely, *tumours of the inguinal region.* In the male, tumours which have descended into the scrotum must be differentiated from the numerous and important group of *scrotal tumours.* As a matter of fact, complete scrotal herniæ bear a close resemblance to very few scrotal tumours. They will be discussed in the separate chapter devoted to this subject.

The following conditions are most apt to cause errors in diagnosis of inguinal tumours:

The tumour is composed of small nodules and strands, which give the impression of an epiplocele. It may be pressed inward and, to all appearances, reduced. Examination of the canal shows that a prolongation of the tumour projects into its opening. In this case everything depends upon the impressions produced by palpation. An experienced touch will decide whether the structure is omentum or not. For we may be dealing with a lipomatous tumour—either with the so-called adipocele or with a lipoma simulating a hernia.

The points of difference between the two are as follows: A fat hernia (adipocele) boasts of a hernial sac. If this is opened its cavity is found empty, but its posterior wall is crowded forward by a lumpy mass, which is contained in the properitoneal space and is immovable. A fatty tumour simulating a hernia is a lipoma, situated in the neighbourhood of the hernial opening.

Sometimes it grows about an empty sac, the fat extending back to the peritoneum.

I remember a case of very soft and finely nodulated inguinal lipoma which exceeded the size of two fists, extended into the scrotum, and covered the external inguinal ring. It gave the impression of an omental hernia, for it was composed of elongated lobules which I mistook for omental strands. One important point, however, caused me to leave the diagnosis in suspense, and to mention the possibility of a fat hernia. This was the presence of several small fatty masses immediately beneath the skin and not directly attached to the tumour.

The following is another condition met with: A small rounded mass is felt in the inguinal canal. It is moderately tense, and gives an impulse on coughing. This impulse, it must be mentioned, is not very clearly marked. If we neglect to examine the scrotum in such a case of cryptorchism, the testicle might be confounded with a hernia. It is therefore necessary to examine the scrotum in all cases of inguinal tumours. Careful examination of the suspected structure, after relaxing the abdominal wall, and the peculiar sensations experienced by the patient, prove that the tumour is the testicle, which is wanting in the scrotum. Curiously enough, it may happen that the cord descends into the scrotum, bends upon itself, and returns to the testicle, which is either in the inguinal region or abdominal cavity. The processus vaginalis peritonæi may reach into the scrotum and contain serum, so that a peculiar variety of hydrocele is formed, although the testicle is above the external ring. Finally, an undescended testicle may be complicated by a hernia. This condition grows especially interesting if the hernia becomes strangulated.

If the undescended testicle has undergone changes in its size and shape through inflammatory processes or malignant degeneration, the resulting tumour may be very obscure. Professor Szymanowsky suffered from cancerous degeneration of a cryptorchid, and died of metastatic recurrences, although the primary growth had been removed by Pirogoff. If we bear in mind that undescended testicles are prone to malignant changes, and that, according to English, prolapsed ovaries frequently are diseased, it becomes a matter of course to suspect these conditions under the above circumstances. The diagnosis of these cases will, however, always remain very difficult.

At times malignant changes in an undescended testicle are accompanied by a serous effusion into the tunica, so that the degenerated testicle is surrounded by an hydrocele. As the effusion usually is moderate, the thickened testicle may be felt through the layer of fluid. As a rule, the corresponding lymph glands are involved. They must be palpated from in front through the anterior abdominal wall, as they are situated retroperitoneally in the vicinity of the kidney.

Another confusing case is the following: The tumour is hard, circumscribed, painful, irreducible, but movable; the testicle is in its normal site. This applies to small herniæ of the omentum if they become inflamed through pressure of a truss or as the result of other traumata. The *inflamed omentum* grows so hard, that unless the condition has been observed on some previous occasion, it is scarcely credible that omentum can acquire such a stony consistency. If the patient is kept quiet, the swelling subsides in the course of a few days, and the hernia can be reduced. The diagnosis can usually be made by aid of the marked mobility of the mass. A pedicle can be felt which moves with the tumour. Usually at some previous time intestine formed part of the hernial contents, as the symptoms mentioned by the patient show.

The femoral region is also occupied by tumours, which require consideration when the diagnosis of femoral hernia is studied. Other pathological conditions must be looked for in this neighbourhood. The lymph glands about the crural canal are numerous, tumours of the blood-vessels occur, abscesses point here—therefore the conditions which have at least one or more symptoms in common with herniæ are very numerous.

With what tumours can a femoral hernia be confounded? In the first place, with an inguinal hernia. If the inguinal hernia has descended into the scrotum or labium majus, the first glance suffices to enlighten us. But if the hernia reaches no farther than just beyond the external ring, a beginner may well pause in doubt.

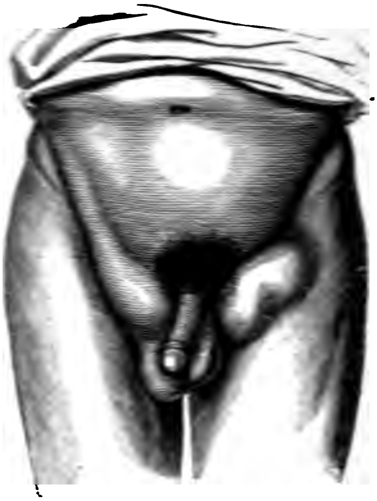

Fig. 29.—Right inguinal, left femoral hernia.

Astley Cooper stated that the position occupied by the hernia, in relation to the spine of the pubis, is conclusive. A hernia situated above and internal to the pubic spine is of the inguinal variety; the femoral hernia is below and external to the spine. Linhart, also, praises this landmark, and claims that it never deceives. In easy cases, it is unnecessary to search for the spine;

inspection shows whether the swelling is below Poupart's (femoral hernia) or above the ligament (inguinal hernia). But difficult cases have been observed, in which inguinal herniæ have taken an abnormal course, extending into the femoral region instead of occupying the scrotum. Similarly, femoral herniæ have been known to reach upward and inward to the external ring or labium. In such cases, reduce the hernia and observe whether it reappears from the crural or inguinal ring. If irreducible, let the patient cough, and watch for bulging along the inguinal canal. If this symptom is not well marked, palpate the crural ring to the inner side of the femoral artery; grasp the tumour, and try to demonstrate a pedicle, which enters the canal. Try to crowd the tumour away from the opening. A similar examination of the inguinal ring should be made with one finger introduced into the ring. The discussion of such rare varieties of femoral hernia as the hernia of

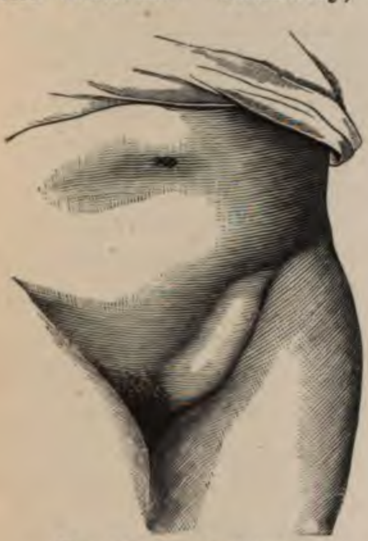

FIG. 30.—Inguinal hernia.

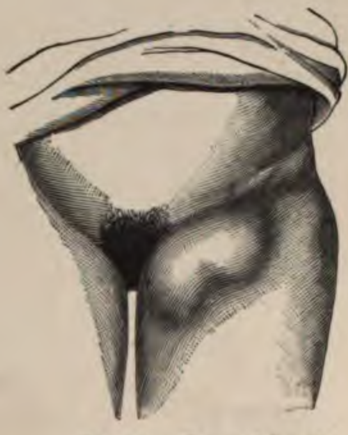

FIG. 31.—Femoral hernia.

Cloquet, of Langier, etc., which are impossible to diagnose in vivo, may as well be omitted.

The diagnosis of *obturator hernia* is also difficult. This hernia is separated from a femoral hernia by the pectineus. Pain radiating along the inner side of the thigh down to the knee, or even down to the great toe, should lead us to suspect this form. Obturator hernia is situated internal to the femoral opening.

Among the other swellings, of non-hernial origin, which occur in this region, the following deserve mention. We naturally assume that the tumour does not give a tympanitic note, and is of small size. *Varices* of the saphenous or femoral vein disappear upon the slightest pressure without a gurgling sound, but reappear at once. Their colour is bluish, and they occur in conjunction with varicose veins along the thigh and leg. A beginner is impressed by the fact that they enlarge during crying or coughing efforts. In order to demonstrate beyond a doubt that the swelling is a varix, it is only necessary to have the patient assume a prone position and to compress the region *above* the tumour. After prolonged, firm compression, the varix increases in size, if the artery is not compressed at the same time. To make the demonstration even more striking, continue the compression above the tumour, and slowly draw another finger, from below upward, along the vein, thus emptying its contents into the dilatation, which, of course, increases in size.

An enlarged, deep-seated inguinal *lymphatic gland* may require a more detailed examination, chiefly because it can be pressed down into the deeper tissues, and consequently simulates the reduction of a hernia. Try to grasp the mass between two fingers; then let the patient cough. The tumour does not increase, nor does it communicate an impulse to the palmar surface of the finger. If the finger is closely applied to the belly wall, its dorsal surface may receive an impulse, which gives a false impression. This should be avoided. Not infrequently, the lymphatic gland can be isolated so that the fingers reach beneath it.

A *psoas abscess*, appearing from beneath Poupart's, may deceive a tyro. The swelling is reducible, but the reduction differs from that of a hernia in that the contents do not glide back suddenly. It has an impulse on coughing. Fluctuation is present above Poupart's, and is communicated to the lower portion of the abscess. In addition, the pathognomonic position of the hip is present, and also the kyphosis of the spine. The *cysts*, which occur in the inguinal region, may be distinguished from herniæ by the absence of impulse, and by their translucency. *Lipomata*, which may extend as far as the peritoneum, are recognised by absence of expansile impulse if compressed from the side.

UMBILICAL HERNIÆ can be mistaken for no other tumours except *ventral herniæ*, which originate close to the site of the navel. The matter scarcely deserves mention, yet it gives an opportunity to practise logical deduction in making a diagnosis. It is possible to find a weak spot in the abdominal wall contiguous to the umbilicus, through which a hernia has prolapsed. It behooves us to decide whether the opening is formed by the umbilicus or by an abnormal gap in the wall. The following signs may be relied upon: 1. The true umbilical hernia is spherical; the ventral elongated. 2. In ventral hernia the umbilical scar is laterally placed, and not in the middle of the tumour. 3. A strictly circular opening is in favour of umbilical hernia.

A rare condition—*hernia of the umbilical cord*—requires only casual mention. Large herniæ are unmistakable. They may contain part of the liver, which is readily recognised by palpation. They increase in size during expiration, and grow tense during crying. Small herniæ of the cord may be overlooked. It is necessary, therefore, to examine the cord of the newly born for small reducible tumours.

Herniæ which appear at rare sites (such as ventral, obturator, sciatic) must be recognised by the general diagnostic signs common to all herniæ.

Diaphragmatic hernia rarely causes symptoms unless it becomes incarcerated. If symptoms of internal obstruction appear, this condition should be borne in mind. After injuries which might cause rupture of the diaphragm, herniæ certainly should be thought of; but, as a matter of fact, they are usually first recognised at autopsy. A large hernia may cause symptoms pointing toward the nature of the trouble, such as dyspnœa; unilateral compression of the lung, with stomach tympany on the same side of the thorax; displacement of the heart; change in position of liver dulness (for the stomach usually helps to form part of the contents of a diaphragmatic hernia). Manual examination of the abdomen by the rectal method of G. Simon might be employed both to clear the diagnosis and to aid in reduction of the hernia.

San Francisco, Cal.

CHAPTER XXIII

STRANGULATED HERNIÆ, AND CONDITIONS SIMULATING INCARCERATION

I HAVE often heard surgeons praised for their boldness, their successes, and their courage in accepting risks. I would like to confront such a surgeon with a patient presenting the symptoms of obstruction, who at the same time possessed one or more irreducible herniæ. Would the surgeon have the courage to say, "None of these herniæ are strangulated; I shall not do a herniotomy"? It requires no courage to operate such a case at the present day; but to refrain from doing a life-saving operation calls for courage of conviction, which is justified only by most careful observation and accurate analysis. The case just presented is, therefore, one in which the thoroughness and experience of an operator is put to the test. Even if these qualities are present in the highest degree, cases arise in which it is impossible to decide whether the symptoms of obstruction are due to the irreducible hernia or to other causes. Translated into a rule for practice, the sentence will read: *In doubtful cases, operate.* The greatest herniotomists have erred, and cut down on non-strangulated herniæ; the most experienced surgeons have failed to discover strangulated herniæ, or have failed to recognise them.

In the present discussion, let it be emphasized, the signs used for diagnosis apply only to typical cases. In doubtful cases, *in dubiis—nulla libertas*, but herniotomy. The diagnosis of incarceration is assured by the symptom-complex of the disease.

First of all, I call attention to the following fact: No experimenter or histologist has demonstrated that strangulation is accompanied by "inflammation" of the affected coil of intestine. Only isolated clinicians spoke of inflammation in strangulated hernia. If the pedicle of a tumour is tied off, or if a finger is surrounded tightly by a string, the parts distal to the ligature do not become inflamed; *stasis* and *gangrene* result. If the unprejudiced observer studies the strangulated loop, with the above-mentioned points kept in mind, he will not discover symptoms of inflammation. The loop will appear red, chestnut-brown, hemorrhagic in spots, gray at the point of strangulation or kinking, or, at a later stage, collapsed, pulpy, gangrenous. The intra-abdominal portion of the gut contiguous to the gangrenous spot may become inflamed; when the loop becomes gangrenous, the sac and all the coverings of the hernia also rapidly inflame, so that abscess formation takes place. The strangulated parts, however, show only stasis or gangrene. Consequently, division of strangulation into periods of hyperæmia, inflammation, and gangrene is unjustifiable.

Two main questions remain for us to decide: 1. Is the hernia strangulated? 2. Has gangrene begun?

The signs of STRANGULATION may be divided into local, abdominal, and general.

Local Symptoms of Strangulation.—The hernia is no longer reducible, although the patient previously was always able to reduce it; reduction now becomes impossible, even to the physician. At the same time, a marked *increase in tension* is noticeable in the hernial tumour, if its contents be gut. We shall limit the present remarks to such cases. This tension increases more or less rapidly from hour to hour, under our eyes. The tumour is sensitive, especially at its neck and more particularly in the vicinity of the hernial orifice (in the inguinal and femoral varieties); deep pressure causes considerable pain. The orifice is no longer patent to the finger, and in some cases a sharply defined constriction of the hernial mass may be recognised at the opening. If intra-abdominal pressure is suddenly increased, no impulse can be felt, for the hernial orifice is entirely shut off. The percussion note is flat.

The flat note obtained over strangulated herniæ may be explained in the following ways: With rare exceptions, strangulation is due to elastic constriction—i. e., the loop is forced through the hernial orifice by the *vis a tergo*. Its contents is milked out, and remains behind. The empty loop is placed outside the orifice, later its lumen is filled with blood and mucus; hence the flat note.

The *abdominal symptoms* which may be looked for are the following: The abdomen gradually distends, owing to the slow development of *meteorism*. Soon after the stasis of intestinal contents has begun the gut contracts intermittently, in a vain attempt to force the fæces past the point of strangulation. New impulses continually stimulate the intestines, which contract with great vigour. Single loops become erect, visible through the anterior wall; in other words, the portion of the *gut situated above the obstruction shows peristalsis*. This overaction, however, does not occur unaccompanied by pain; on the contrary, *severe, spontaneous pain, colicky in nature*, is felt in the hernia, and, more especially, radiates about the navel. At first the belly is merely sensitive to the touch; later, pressure causes pain. Some time after the strangulation has taken place the patient begins to *vomit*, not violently, as a rule. At first the vomitus consists of stomach contents; it then becomes greenish and bitter from the admixed bile; and toward the end, feculent intestinal contents, or, in some instances, a true fæcal vomiting, appears. Constipation is absolute. As this symptom requires considerable time to develop, another earlier sign must be relied upon. This is the *inability to pass gas*, in spite of repeated and violent efforts.

The *general symptoms* are not of particularly striking nature. At first the patient suffers from malaise; he then grows anxious, and his pulse becomes rapid. Frequent vomiting tires and weakens him still further; his extremities grow cold; the pulse small, rapid, and intermittent. The facies is found altered, the nose is now sharp, the eyes sunken, the skin cadaverous in colour, and covered with cold sweat. Unless exceptionally strong, the sufferer dies of exhaustion and intestinal auto-intoxication. Only few live to be relieved by gangrene of the affected loop, with external rupture and spontaneous cure by the formation of an artificial anus. The development of this outcome can be diagnosed only by the local signs. Marked œdema and inflammatory redness of the skin covering the tumour, swelling of all the layers composing the hernia, which grows broader and flatter, point to it. The peculiar crepitation obtained over the swelling shows that the deep-lying structures have become gangrenous, and that suppuration is taking place. *Gangrene of the strangulated hernia is, therefore, recognised by the occurrence of a phlegmon of the hernial coverings, accompanied by the usual signs of suppuration.*

The various symptoms of strangulation preserve a certain sequence and quantitative relation to one another. At the outset, when the tension in the hernia is not yet very great, meteorism is moderate, and vomiting occurs spontaneously, at intervals of one or two hours only, unless the patient drinks (as his ever-increasing thirst constantly tempts him to do). Drinking is promptly followed by vomiting. The general condition is but slightly disturbed. If the obstruction is unrelieved, the tumour increases in size; the colicky pains become more violent, the belly more distended and painful, spontaneous vomiting increases in frequency, the pulse rate is accelerated. This sequence and relation of the symptoms may be termed *harmony* of the signs of strangulation.

The rapidity with which this symptom-complex develops depends upon manifold reasons, many of which are but partly understood. Cases have been observed in which strangulation caused death within a few hours. In these, vomiting was incessant, pain very marked, and exhaustion rapid. In other cases the course is slow; the strangulation persists for many days, even longer than one week, and all the symptoms develop with corresponding slowness. In most instances we may take for granted that the danger of gangrene is very imminent toward the end of the second or beginning of the third day. The course may be classified as *peracute*, *acute*, and *subacute*. Certain local findings at the site of the hernia are of more value than the amount of pain or the frequency of vomiting. If the hernia is very tense, the neck thin, the hernial canal narrow (inguinal, or, in the female, femoral), the constriction tight, gangrene will occur more rapidly. Local conditions, less constricting in nature, will produce a more subacute course, and not demand as early surgical interference. Rarely, cases are observed in which, in spite of persistent strangulation, vomiting does not appear. The other symptoms, such as tension in the hernia, irreducibility, local pain, and absolute constipation, are, however, present. Be guided by the local symptoms in these cases, and operate. I was greatly surprised, when operating upon an incarceration of five days' duration (unaccompanied by vomiting), to find typical strangulation.

Exceptionally one or more symptoms may vary or be absent in cases of true strangulation. Although constipation and vomiting stand at the head of the list, among the symptoms of strangulation, the bowels may, in exceptional instances, move during the attack.

In the first place, the movement may take place by emptying of the segment of bowel situated below the point of obstruction. This usually follows the administration of an enema. In the next place, the strangulation may be of such a nature that the lumen of the bowel is not totally obstructed. If the vermiform appendix or a congenital diverticulum of the small intestine forms the hernial contents (Littré's hernia), it seems possible that the fæces should not be completely obstructed. But, even in these conditions, constipation has been observed. In an oft-quoted case of Dieffenbach's, choleraic diarrhœas were noted. Goyrand quotes a similar case. Dieffenbach, who performed more than six hundred herniotomies, mentions two cases in which, although vomiting was wanting among the symptoms of strangulation, stupor appeared. Both cases were in old men. In the one, operation showed a congested and strangulated loop. After the constriction was relieved the patient awoke from his stupor. Cases in which the abdominal pain is insignificant are not uncommon. Strong, healthy peasants do not complain of pain; they may walk long distances in order to seek medical advice for a "spoilt stomach." One case came to my knowledge where an exceptionally robust fellow undertook an hour's journey to seek his physician. He had been vomiting for a whole day, the strangulation was severe, and yet he had shown no signs of weakness. Attempts at taxis were made with the patient in the erect posture. Only after these had been continued for some time did the patient drop down in a faint, and then reduction promptly followed.

If merely a portion of the intestinal wall is strangulated the lumen may yet be completely occluded, for the two segments of the loop usually lie parallel, and therefore the severest incarceration may result. A hernia of the appendix, or of Meckel's diverticulum, may have advanced to gangrene without producing complete obstruction.

All of the preceding goes to show that in an atypical course of strangulation the local symptoms—*irreducibility, tension*, and *pain at the hernial orifice*—are of the utmost importance. The tension, especially, is significant. Danzel, who has had great experience, calls the tension "characteristic." It is indeed so, but the peculiar sensation imparted to the finger can not be described in words; the hernia must be palpated. This must include the answer to the question of how to determine *the* incarcerated hernia, if symptoms of strangulation occur in an individual who has several irreducible herniæ.

We now come to the question of how to act if symptoms *simulating strangulation* set in, especially absolute constipation, vomiting, and abdominal pain. Patients are brought to a medical clinic with supposed peritonitis, and a strangulated hernia is discovered. Or, just as frequently, patients who are suffering, not with strangulated hernia but with peritonitis, are brought to the surgeon for taxis or herniotomy. The first thing to do in all cases is to examine the usual hernial sites, and to determine whether or not there is a hernia; and, if so, whether or not it is strangulated. A cursory examination will not suffice, for not only should the more common sites, such as inguinal, femoral, and umbilical regions, be examined, but the possible occurrence of sciatic and obturator hernia must be kept in mind. If these regions are found negative, the whole abdomen must be examined, in order to exclude a ventral hernia which might occupy any situation. It is evident that a small hernia may readily escape notice if the opening is deeply situated and the patient fat. But, as even a *small* strangulated hernia is tense, has a smooth surface, and is painful at the hernial orifice, it ought not to be overlooked. In the Allgemeine Krankenhaus, in Vienna, a patient was admitted for strangulated ischiadic hernia, because a tympanitic note was found over a certain spot in the gluteal region. I mention this case in order to bring out the point that a tympanitic note is always obtained over the sciatic foramen unless the individual is specially fat. Therefore this sign can not be relied upon. If, however, the hernia is larger, the tumour can not be overlooked.

If no tumour is discovered, *internal strangulation* is by no means excluded.

Let us assume that we are dealing with such a case, and attempt to rule out other conditions simulating internal strangulation. The first condition requiring exclusion is PERITONITIS. The chief points of difference may be thus tabulated:

1. In obstruction, the patient moves, gets out of bed, and walks about. In olden days such patients were forced to dance. In peritonitis, the sufferer lies in bed and avoids all motion.

2. Spontaneous pain, especially colic, is characteristic of obstruction; pressure causes pain only at the site of strangulation. In peritonitis, the slightest pressure causes pain; the whole abdomen is sensitive.

3. Obstruction is accompanied by violent peristalsis; the loops of gut squirm and grow erect; peristaltic waves move toward the point of stenosis. Peritonitis early causes intestinal paresis.

4. Obstruction runs an afebrile, peritonitis a febrile, course.

We see cases of intestinal obstruction in which a general peritonitis develops at an early stage. In these cases the more important peritoneal symptoms rapidly assume prominence—especially the general tenderness to even slight pressure and the fever. Here the constipation and vomiting are often ascribed to peritonitis. Even in such cases the diagnosis should be directed into the correct channels by the vomiting, which resists all treatment, constantly grows more severe, and finally becomes fæcal. It might be added, that the most important symptom of peritonitis is the demonstration of an inflammatory exudate collected in the free peritoneal cavity. But just in these cases it is rarely possible to obtain dulness, for the exudate lies hidden among the distended loops of obstructed intestine. The inflammation must be recognised by the pain on pressure and the fever. On the other hand, the uncontrollable vomiting, especially fæcal vomiting, conjoined with absolute constipation (both inability to pass fæces and gas), serves to distinguish obstruction from peritonitis, be its origin rheumatic or perforative. The course of obstruction differs particularly from the last-mentioned variety of peritonitis. Perforation produces a strikingly rapid collapse, while peritonitis, due to intestinal obstruction, has as its prodromal symptoms colicky pain, vomiting, *followed* by distention, tenderness, fever, and again and constantly, vomiting. The duration covers many days, but in these cases surgical interference can do but little good. That success can be expected in *internal strangulation*, not accompanied by peritonitis, must never be forgotten, for we are fortunately able to recognise the condition in many instances. The onset is slow and afebrile. Colicky pains and tympanites are the first symptoms. The patient, unaware of his grave condition, tries to effect a cure with household remedies. Whisky and bitters, a diaphoretic, a long walk, and a cathartic are tried in turn. The last produces vomiting, but the bowels refuse to move. The patient ascribes it all to a "spoilt stomach." The colic and vomiting continue; the constipation is unrelieved. Finally, the physician is called in. "Woe betide him," says Niemeyer, "if he has failed to examine carefully for a possible hernia." We assume that he does not deserve this reproach; that he has minutely examined the belly; that he has explored the rectum for fæcal impactions and other obstructions. Nothing is found. But constipation, meteorism, vomiting, can not lightly be explained away. The unmistakable picture of obstruction rapidly unfolds itself with all its sinister concomitants—a picture which Watson and Niemeyer have described classically. Such patients suffer great torture. Their abdomen grows more and more distended, till it assumes the shape of a barrel. The diaphragm is pushed upward; the intestines are seen through the abdominal wall twisting and squirming. Each rising and falling peristaltic wave produces rumbling and rolling, with colicky pains. These efforts are all in vain; the intestinal contents can not pass down, and fæcal vomiting sets in. At this stage the patient's appearance is appalling; his pulse grows small, and death, from exhaustion, finally comes to his relief.

The beginner must be taught, from the outset, that not infrequently peritonitis runs its course with severe vomiting and obstinate constipation, sometimes unaccompanied by fever. Occasionally purulent peritoneal exudates produce this picture along with an astonishing degree of euthanasia. But the very condition of euthanasia, when combined with constant vomiting and *moderate* distention, serves to distinguish peritonitis from obstruction. In obstruction, ceteris paribus, tympanites is very marked when vomiting has grown continuous.

The actual cause of obstruction can usually not be determined. *Invagination* may be suspected if gas or a small amount of fæces, accompanied by blood, are passed. The invagination may positively be recognised if rectal examination allows the finger to reach the obstruction, or if the invaginated gut appears at the anus. While a student at Oppolzer's clinic, I saw a case in which a piece of the intestine sloughed off, and was found in the stool.

Other conditions producing obstruction may, for the sake of convenience, be grouped as follows: 1. Causes of obstruction *outside* the intestine; among these are strangulation by omental bands, adhesions, holes in the mesentery and omentum, incarceration in diaphragmatic hernial openings, and, finally, in normal fossæ, such as the subcæcal fossa, through the foramen of Winslow, etc. 2. Cause of obstruction *situated in the wall* of the gut, such as volvulus, knots, stricture, especially carcinoma of rectum. Both varieties may be acting at the same time—as, for instance, in volvulus, in which the mesentery of the affected loop may simultaneously compress another loop of gut. 3. Cause of obstruction *within the lumen* of the intestine: here may be mentioned obstruction due to undigested food, fæcal concretions, gall-stones, foreign bodies, ascaris, etc. This variety of obstruction may occur in a loop of gut within the sac of a large hernia of long standing. Larrey operated on a hernia in which the obstruction was due to ascaris, and after the worms had been moved on by massage the symptoms of strangulation were relieved. This, and other similar symptoms, go to prove that there is a kind of obstruction found in herniæ which closely corresponds to the "fæcal incarceration" of the elder Richter.

It is readily understood that in a given case, in which the diagnosis wavers between obstruction and peritonitis, a single loop of intestine, which is felt at the same spot at repeated examinations, may be an important landmark and aid. This is the rightly famous *fixed loop*, which not only decides in favour of obstruction, but also points to its site.

The scope of this book forbids a detailed discussion of the method of diagnosing the variety and site of an intestinal obstruction. In some cases the concurrence of a number of factors makes it possible to arrive at a diagnosis; in other cases the cause and the site of the obstruction remain in doubt. For instance, a patient has suffered for some time from constipation, occasional vomiting, and meteorism. All of these symptoms are increasing in severity, the fæcal masses approaching more and more to the type of lead-pencil stools. In addition, the patient, who is somewhat advanced in years, notices an increasing cachexia. The diagnosis of *Carcinoma* must be entertained, especially if palpation detects a resistance, rectal examination reveals a stricture, or a tumour of the abdomen can be found. V. Dumreicher was called in to treat a case of incarcerated umbilical hernia. Upon examination, he found a carcinoma of the transverse colon, which was about to perforate.

Another case may occur without prodromal symptoms. For instance, a patient suffering from acute obstruction has had a herniotomy performed upon him at some preceding time. At the time of operation adherent omentum was found at the entrance of the hernial canal. Our first suspicions will lay the cause of the present strangulation at the door of adherent omentum. If the patient has previously had peritonitis, and now suffers from acute obstruction, we are justified in suspecting strangulation from pseudomembranous bands. A history of persistent constipation will confirm this theory, for the kinking of adherent coils would readily account for the sluggishness of the bowels.

The site of the obstruction may often be recognised by the *distribution of the meteorism.* Distention of the small intestine (rounded belly, the region of the colon not distended) points to an obstruction above the large intestine. If the tympanites is insignificant in amount, look for the obstruction at the beginning of the small intestine. A generally distributed distention points to obstruction near the end of the colon. The examination per rectum by means of the entire hand, after Simon, is of especial value. As Simon has shown, the fingers may reach well into the sigmoid, and if this is raised, the belly may be palpated still higher up, even as far as the ensiform. By this method a constricting band or a hard mass may often be reached.

Sometimes an area of increased resistance may be felt by abdominal palpation. At this spot motion is less noticeable, in spite of increasing tympanites, than in the rest of the belly, through which peristaltic waves travel to and fro. The pain may have started here and have persistently remained at this site ("fixed loop").

We may hope to receive some information by injection of water through the rectum or distention of the intestines with gas. These measures, in conjunction with the other symptoms, may be of great assistance. It is evident that the obstruction may be looked for low down (let us say in the flexure), if repeated injections of water fail to force more than a small quantity into the bowel.

The above-mentioned conditions will sooner or later (depending upon the experience or inexperience of the surgeon) allow us to recognise an internal strangulation or obstruction. In an irreducible hernia, accompanied by constipation, vomiting, and abdominal pain, the conditions are different. We are here confronted by a positive, unmistakable indication—operation for strangulated hernia. The fear of cutting down upon a non-strangulated hernia restrains us from too hasty interference. The dilemma is increased if the tumour is not positively a hernia, for, by operating, we then incur the blame of having confounded a lipoma with a rupture.

We are mainly interested in finding out whether, in a given case, it is possible to say that no strangulation exists. In certain cases strangulation may be positively excluded.

Let us assume that a hernia of long duration, which had always been reducible, suddenly, for some reason, grows painful and irreducible, with the simultaneous onset of vomiting. We find an undoubted case of scrotal enterocele, for the tumour gives a tympanitic note. The tension and pain on handling are quite marked. Taxis can not be attempted on account of the tenderness. The patient receives an injection of morphine, and is placed in a proper position. Sleep follows, and next day we are informed that the vomiting has ceased and gas has been expelled. Renewed examination of the tumour shows it sensitive at all points except in the vicinity of the neck, and pain is entirely wanting *above* the external ring. A fine crepitation attracts our attention during this examination; this crepitation resembles the "feeling produced by squeezing soft snow." The same crepitations have been observed in other conditions—for instance, after injecting a hydrocele with tincture of iodine—and are due to an adhesive INFLAMMATION. The inflammatory exudate which covers the smooth serous surface produces the crepitant sensation. That this same process takes place on the serosa of the intestine and of the hernial sac is abundantly proved by the sacs encountered, some of which are adherent to the gut over its entire surface.

At times the adhesions form without producing any inconvenience; at other times, vomiting and pain in the hernia accompany their formation. The vomiting is not surprising, for, according to the researches of Claude Bernard and Brown-Séquard, we know that irritation of the peritoneum, abdominal wall, and abdominal viscera can cause vomiting. And, in our suppositilious case, the patient vomits after the examination, but this does not disturb us. We have demonstrated inflammatory products within the hernial sac. Pain, limited to a given point—the point of strangulation—is conspicuous by its absence; the pain found is distributed over the entire tumour. We can safely wait. During the course of the same day the bowels move; the abdomen does not grow distended, and diffuse abdominal pain does not appear. The general condition remains good, but occasionally, say once a day, the patient still vomits. Gradually the crepitations within the hernia become less marked, vomiting does not recur, appetite and bowels become normal, and local tenderness is no longer felt. The hernia, however, has changed from the reducible to the irreducible variety.

In another case, the symptom-complex is identical, except for the local findings in the hernia. The contents in this case is omentum. Instead of crepitus, a disproportionately rapid increase in size occurs; the tumour fluctuates, and is translucent. Here, evidently, a rapid *effusion* into the hernial sac has taken place. If, in the preceding case, the inflammation was adhesive, in the present instance it is *exudative*. Just as the physician diagnoses an adhesive pleurisy by the pleural friction râles, a pleurisy with effusion by the flatness, so the surgeon in these cases recognises the adhesive inflammation by the friction, the exudative by fluctuation and by the light test. In neither of these cases was the intestinal tract obstructed. The belly was neither distended nor painful; the vomiting did not increase in the ratio in which it should if the local, abdominal, and general symptoms were due to intestinal obstruction. Certain contradictions which do not correspond to the picture seen in obstruction are present.

The following is a different case. The conditions are irreducible hernia, constipation, and vomiting. We are surprised by the marked distention of the abdomen, the great care with which the patient avoids all movement, the superficial respiration, and the excessive tenderness of the entire belly. In marked contrast, the hernia is soft, and not tender; the abdominal and local symptoms disagree. If no hernia were present, we would at once make the diagnosis of PERITONITIS. The history shows that the patient has had a chill on the preceding day, and that the abdominal pain was severe without pain in the hernia itself. Gas was passed. The temperature now is 38.5° (101.3° F.). This is not the picture of obstruction. We again wait and observe. The vomiting does not recur on the same day, and on the following day free exudate can be demonstrated in the peritoneal cavity. This is the picture of an acute *diffuse* peritonitis. If the peritonitis is *circumscribed,* the symptoms of some other disease precede it—the symptoms of appendicitis, gastric or duodenal ulcer, or perforation of tuberculous or typhoid ulcer of the small intestine. We always find an entire absence of local symptoms except the irreducibility of the hernia (which we have assumed to be present from the outset), and positive symptoms elsewhere. These consist of marked general abdominal tenderness, fever, and, at a later stage, free exudate, *in general*; and severe local tenderness, fever, and local dulness or flatness *in circumscribed* peritonitis. The process sets in not with pain in the hernia, but with pain in the abdomen. In peritonitis, the vomiting stops, and at least gas is passed; in obstruction, the vomiting increases, and becomes fæcal.

We may find another class of cases in which, although a tumour is found at a hernial orifice, the nature of the tumour is uncertain. Vomiting and constipation are among the symptoms. The condition is known as PSEUDO-STRANGULATION. A strangulated *testicle* illustrates the point. It may happen that an undescended testicle within the inguinal canal grows inflamed and swollen, or that the testicle, as the result of some trauma, slips up into the canal, and then becomes painful. The scrotum should, therefore, be examined in all cases, in order to avoid overlooking a possible cryptorchism. As a rule, vomiting occurs, but does not increase in severity, and never grows fæcal; moreover, the abdomen remains soft, and both gas and fæces are passed. Cases, however, are on record in which vomiting (even fæcal vomiting) and constipation were present, the operator being thus compelled to expose the tumour. This is entirely justified, especially as a hernia may be incarcerated above the undescended testicle.

Inflamed *inguinal glands* produce a tense, painful, and irreducible swelling, accompanied by the symptoms of vomiting and constipation. As the local symptoms are severe, we are more easily influenced to decide in favour of herniotomy, and when we cut down, find—inflamed glands. Our herniotomy has changed into an extirpation of the glands. Many such cases are on record.

Knowledge of the above occurrence has in some cases, proved fatal to the patient. It happened to Laharpe that his herniotomy incision laid bare glands, and he at once assumed that the symptoms were due to peritoneal irritation. The autopsy showed that behind the glands a small hernia was incarcerated. This experience has been duplicated by several other surgeons; for instance, by Danzel.

Danzel has shown that *inflammation of an empty sac* causes symptoms resembling strangulation. In these very cases an error is more readily committed, for a tumour is found, which, according to the patient's history, points to the previous existence of a hernia.

In all these cases the axiom, that operation is indicated if constipation persists and vomiting increases, holds good. If no hernia is found, usually some other condition will be relieved; if strangulation is found, the patient has been saved. Strohmeyer said to his pupils: "If you find a strangulation during the day, it should be relieved before sundown; if during the night, it should be relieved before sunrise."

CHAPTER XXIV

DIAGNOSIS OF THE LAYERS AND ANOMALIES ENCOUNTERED DURING THE OPERATION OF HERNIOTOMY

MALGAIGNE'S advice was, Cut down until you can cut no farther, in doing a herniotomy; and the present chapter will prove to be nothing but a paraphrase upon this theme. The implication is to cut down till you reach the gut, but not to injure it, whatever else you do. The problem then narrows down to the query, How do you recognise the gut? Saviard, who lived toward the end of the seventeenth century, and was surgeon at the Hôtel-Dieu in Paris, discussed this subject. Pitha treated the same theme in a masterly manner; and a tried practitioner—Pauli—who died only a few years ago, left a monograph which enters fully into all the details of the question. But, as on the whole the gut can be mistaken only for the hernial sac, the matter narrows down to two questions: 1. How do we recognise the sac? 2. How do we recognise the gut? This division is important, for the young operator who does his first herniotomy will pause in doubt when he reaches the sac. Linhart, following the example of Astley Cooper, calls all the layers lying between the superficial fascia and the sac, fascia propria, while Cooper introduced this name only in femoral hernia. All the layers, no matter whether they originally were of connective tissue or muscular origin, together form one stratum, which can, however, be split up into many planes. This stratum is recognised by the fact that, after cutting through the superficial fascia, it is exposed as a well-defined and circumscribed covering of the hernia. The superficial fascia accompanies the skin edges of the wound when these are moved to and fro. Moving the skin edges does not influence the fascia propria at all. The development of this fascia varies greatly in different herniæ. Compared to femoral herniæ, umbilical herniæ have only a slightly developed fascia. The duration, also, has its influence: a long-standing hernia has a thick, a newly acquired hernia a thin, fascia propria. An experienced herniotomist will bear most of these points in mind in judging a given case, but the tyro need remember only those rules which will help him to differentiate sac from intestine. The following rules should be remembered: After cutting through the superficial fascia and exposing the circumscribed hernial tumour, divide all succeeding tissues, layer by layer. Raise a fold, incise it, and, after introducing a director, cut down upon this instrument. As long as thin, translucent layers can be raised up between the forefinger and thumb of the left hand the gut is in no danger of injury. By following this rule, a layer is finally reached, from beneath which (after it is incised as described above) a thin stream of fluid spurts out of the small opening, or a small tag of fat prolapses. Usually this signals the opening of the hernial sac. Now enlarge the hole until the finger can be introduced, and then enlarge further by cutting down upon the finger. The fluid that escaped proves to be fluid within the hernial sac

(Bruchwasser); the fat that prolapsed was an omental tag. Only exceptionally does this not hold good. The fluid may come from a cyst adherent to the hernia, from a distended connective-tissue gap, or from a hydrocele (encysted hernia). In these cases we find an empty, closed cavity, and the hernia lies more deeply.

In many cases the beginner will hesitate before opening the sac. The round tumour, which lies before him, will make him pause to decide whether it is gut or hernial sac. Let him remember that the intestine is shiny and smooth as glass, and never has any fat upon its surface. The sac is dull, and its surface may be covered with flat clumps of fat. In addition, the gut never has any transparent areas, while they are frequently found on the sac, especially on its dependent portions. If all these means of recognition are insufficient, he must call the sense of touch to his aid. Let him try to raise a small fold; if he can, and if it prove translucent, let him cut through it without further misgiving. But if the sac is thick, as it sometimes is, moving the fold between the fingers will give the sensation of rubbing two smooth serous surfaces together; if, on the other hand, it is gut, the rubbing of the two mucous surfaces imparts the sensation of rubbing velvet. Further, by pressing deeply into the fold, which is grasped by the finger-tips, a tense structure—the gut—is felt within, if the sac has not been completely opened.

Finally, pass the finger upward to the hernial opening. If the gut has been reached—that is, if we are inside the sac—we come to the incarcerating ring; this ring has such a sharp outline that, once felt, it is never forgotten. If still outside the sac, the ring has no such sharp edge, and the smooth coverings all merge into the ring.

There are certain other signs which at once decide the question. For instance, if the tumour has an hourglass form or shows constrictions, nothing but the sac can cause these. Or if, for example, the ring has been incised, and, after reduction of the mass, an empty pocket has been left, this can be nothing but the sac. Again, if the swelling has one or more epiploic appendages on its surface, it certainly is large intestine. The hernial sac is most readily distinguished from intestine if the former is transparent. Dieffenbach, in more than six hundred herniotomies, met with only a single case in which the sac was very thick, but transparent as glass, so that the intestinal loops could be seen through it. Such a pretty demonstration, however, is very rare.

In inguinal hernia, my former assistant, Dr. R. Frank, has recently worked out an astonishingly simple method of orientation.

As sac *and* vas deferens both are contained in the tunica vaginalis communis, an incision which exposes the vas must also lay bare the sac which forms the tumour found next to the vas. In order to reach the cord, which is the guiding structure, without delay, the incision is made into the inguinal canal, and *not* over the tumour. By cutting down into the depths, the vas is reached, and here in the canal the sac can readily be separated from the seminal cord. By this step we prepare for a radical cure after the strangulation has been relieved, for the neck of the sac is dissected free along its entire circumference without opening its lumen. In most cases the hernia may then be reduced without opening the sac, because in the majority of instances the cause of strangulation is more often situated without than within the sac itself. If reduction is resisted, the sac is now opened, but at a more dependent site. This is readily accomplished, for, after the neck has been laid bare, the remainder can be reached by cutting down on a grooved director. This innovation is a great advancement in the technic of herniotomy. Its significance is as follows: Even in dealing with a strangulated hernia, plan your operation as if you were preparing to do a radical cure on a reducible hernia. If this method is followed, no difficulty will be experienced in operating in cases of strangulation.

It is readily understood that this method can be employed in femoral hernia as well. The neck of the hernia, as Gussenbauer's pupils have shown, should be laid bare by Bassini's incision. When the subserous space has been reached and the neck freed, it is pulled from under Poupart's as if from under a bridge. If strangulated, the strangulation should first be relieved by nicking the ring.

It is just as well to know the more ancient rules of classic herniotomy, because they will be found a better guide in atypical cases, for which we should always be prepared.

When no longer in doubt whether the sac has been opened, certain diagnostic tests must also be made upon the hernial contents.

How do we recognise that the loop is or will become gangrenous at some small point? A gangrenous patch is gray, cold, collapsed, and lustreless. A blackish spot will also become gangrenous, and requires precautionary measures on the part of the operator.

The other points of diagnosis will not bear general discussion, for they border upon casuistry. Wattman opened a hernia; fæces flowed out. He at once concluded that he had opened the gut. The string-like process which he felt upon the tumour he diagnosed as appendix, the mass itself as cæcum. He inserted his finger and reached a cleft; this he took to be the ileocæcal valve. His deductions were logical, yet the supposed cæcum proved to be the hernial sac, the supposed appendix a connective-tissue adhesion, and the supposed ileocæcal valve a tear in the prolapsed loop of small intestine. This shows the possibility of error. But, though Skey found omentum and gall-bladder in a femoral hernia, this is not sufficient reason to conclude at once that we have happened upon the most unexpected. Danzel's aphorism, "In every herniotomy prepare for a new experience," hits the mark.

Two points I desire to emphasize. Examine every mass of omentum carefully, for a loop of intestine may be concealed within it. After reduction, the finger should be passed into the peritoneal cavity and the whole circumference of the ring carefully palpated. If it is found free, the finger is withdrawn, and the interior of the sac is now palpated in order to determine the presence or absence of diverticula. If these precautions are neglected, we visit the patient several times in the course of the next few hours, in order to guard against a continuance of the symptoms of strangulation. The cause of further trouble varies greatly. A detailed account may be found in Streubel's classical article, "On Apparent Reductions" ("Scheinreduction"). Danzel says of herniotomy: "The clear and indisputable indication of this measure, its certainty and rapid means of relief, are attributes which make herniotomy a valuable and favourite operation to the surgeon." Though we agree with him, let us take the above-mentioned book and—*nocturna versate manu, versate diurna!*

The following will conclude my remarks: A special form of reduction is *reduction en bloc.* Any one may have the ill-luck to reduce the hernia with its sac, and the strangulation will then continue unrelieved within the peritoneal cavity. Many such cases have been diagnosed and operated upon. The diagnosis can be made from the fact that (1) an irreducible hernia with symptoms of strangulation was found; (2) taxis was difficult; (3) reduction was accomplished suddenly, perhaps accompanied by a tearing sound, and without gurgling; (4) symptoms of incarceration persisted; (5) careful inspection of the abdomen, in cases of inguinal hernia of the scrotum also, shows a retraction (in inguinal hernia the testicle is drawn up); (6) in the neighbourhood of the hernial ring the belly is full and tense. Occasionally coughing will cause the hernia to reappear. If not, incision of the abdomen will show the whole sac in the preperitoneal space. Draw it out, open it, and relieve the strangulation.

CHAPTER XXV

SCROTAL TUMOURS

Make it a rule to determine at the outset from which side a scrotal tumour takes its origin. In simple cases, inspection alone suffices; in doubtful cases, the sound side will enlighten. That side on which only the testicle and its adnexa are found is the healthy one. Whether this is the right or left, can be determined by following up the vas to the inguinal canal. This test may be necessary in some scrotal tumours—for instance, in eventrations—in which the raphe has been obliterated, and the testicle of the sound side pulled or crowded in any direction. This examination will also show whether both testicles are present or not.

The next manipulation is intended to test the limits of the tumour in relation to the external ring. Let us assume that the surgeon stands to the right of the recumbent patient. He should place the four fingers of his left hand beneath the scrotum, and, so to speak, load the tumour upon them; the thumb is placed on the anterior surface of the swelling, close to the external ring. By combined pressure, anteriorly with the thumb, posteriorly with the index-finger, the swelling can be palpated and allowed to glide between these two tactile surfaces. This will enable us to judge whether we are dealing with the vas and its covering or with some other mass in addition, and also whether the vas is normal or abnormal. Let us assume that the manipulation shows that the tumour extends into the inguinal canal, or that it leaves us in doubt whether the spermatic cord alone, or cord and some other structure, are present. This uncertainty is apt to occur if the patient is stout. Two possibilities must then be considered. The swelling either ends somewhere within the inguinal canal or it extends into the abdominal cavity. A tumour which extends into the abdomen may be a hernia, a hydrocele in the open processus vaginalis peritonei, or the so-called bilocular hydrocele. These three swellings have certain points in common. They can be partially or completely reduced, lying down makes them smaller, and coughing imparts an impulse. Reduction, however, is characteristic of each variety. The hernia reduces suddenly, often accompanied by a gurgling sound if the contents is gut. The fluid in the open hydrocele runs out slowly in response to steady pressure, sometimes giving the impression of trickling fluid caused by the currents set up in the narrow neck of the sac. The bilocular hydrocele can be only partially emptied toward the abdominal cavity. It is, however, desirable to possess other clear and unambiguous signs by which the correct diagnosis can be made. The following will be of service: A tympanitic note over the tumour points to hernia; translucency excludes hernia. A *hydrocele* of the open *tunica vaginalis* and a *bilocular hydrocele* must next be differentiated. The latter represents a collection of fluid in the tunica vaginalis communis; the contained fluid can spread only in the extraperitoneal pelvic space, and must have a definite boundary within that area. Fluid in the processus vaginalis peritonei reduces into the general peritoneal cavity, and is lost in a relatively boundless space. The above-mentioned variety of bilocular hydrocele takes its origin from an effusion of blood. The true bilocular hydrocele originates from a vaginal process, which is originally shut off above, but which, after the hydrocele sac is overdistended by the accumulating fluid, gradually pushes a diverticulum upward. In this form the fluid is limited within the pelvis by a serous membrane, which is the diverticulum of the closed processus vaginalis peritonæi. The well-defined boundary within the pelvis is characteristic. Consequently, in each case of bilocular hydrocele (called by the French *hydrocèle en bissac*) a circumscribed fluctuating mass can be felt in the iliac fossa. Its contents can be partially emptied into the scrotal portion, and vice versa. Pressure upon the scrotal division increases the tension of the intra-abdominal part, and vice versa. This sign makes it impossible to confuse the bilocular hydrocele with either of the other two tumours. It holds true no matter what the contents prove to be—serum, blood, or even pus. This sign is especially valuable in reference to adherent herniæ which at the time of examination happen to contain no air, for they also are only partially reducible. The impulse on coughing, as has been mentioned before, is more direct and precise in herniæ, but the well-defined circumscription of the swelling within the abdomen excludes hernia beyond a shadow of doubt.

Other scrotal tumours which extend toward the abdomen need not be considered from the standpoint of differential diagnosis in connection with the above-mentioned three forms of scrotal swellings, because their nature can be recognised by other signs. A malignant neoplasm of the testicle, which extends along the vas into the inguinal canal, is recognised by other symptoms. The nature of a *varicocele* is also apparent from both its appearance and its peculiar consistency. At the worst, a varicocele might be confounded with an epiplocele, because both have a cordlike arrangement. But error is avoided by noticing the ready compressibility of the distended veins in varicocele, their increase in size when the proximal end is compressed, the bluish colour of the veins which give the impression of angle-worms, and, finally, the coincident varicose condition of the skin of the scrotum.

If we have demonstrated that the tumour does not extend through the inguinal canal into the abdomen—as can be done by proving inability of reduction, either total or partial failure to empty the fluid, and absence of impulse on coughing—other possibilities must be taken into consideration. The tumour may end within the confines of the inguinal canal. Hydroceles are met with which first distend the scrotum and then end high up in the inguinal canal. If the swelling is more cylindrical, and the circumference of the testicle can be felt free at its lower end, or at least distinctly separated from it, the plainly fluctuating and translucent tumour is a hydrocele of the funicular process. If the swelling is pear-shaped, with its narrower end above, and the testicle hidden, the tumour is an ordinary hydrocele of the tunica vaginalis. In these cases it may be possible to draw the tumour downward away from the inguinal canal, and thus more plainly define its upper boundary.

The typical *funicular hydrocele*—serum in the unobliterated remains of the processus vaginalis—forms a *small* tumour, which not infrequently assumes the shape and consistency of the testicle. The inexperienced may mistake it for a third testicle; but as such an occurrence is unknown, this suspicion may at once be dismissed. The hydrocele of the funicular process is also more translucent and usually more tense than the testicle.

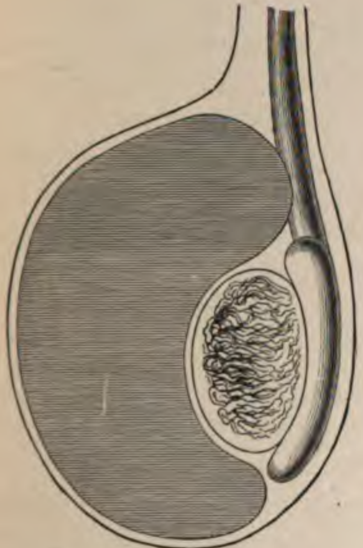

FIG. 32.—Hydrocele of the testicle, or tunica vaginalis (ordinary hydrocele).

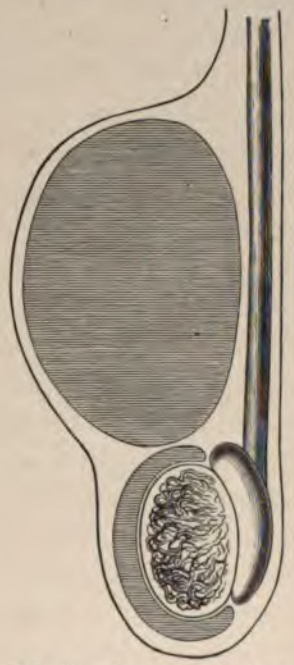

FIG. 33.—Funicular hydrocele.

It is not always possible to demonstrate translucency in a hydrocele, for the sac may be greatly thickened, and in some cases contain plates of bone. Translucency is characteristic of a purely serous effusion. Thickening of the walls points to a plastic inflammation of the tunica, to which the hydrocele owes its existence. Taking all other signs for granted, we then only remain in doubt as to whether the contents is purely serous or hemorrhagic; but a collection of fluid in the tunica always exists under these circumstances. A *hematocele of the tunica* can arise only if a hydrocele has preceded it. The other variety of hematocele—*hematocele of the funicular process*—consists of an effusion of blood into the connective tissue around the cord, which extends into the subserous tissues of the pelvis. This hematocele might be called a hematocele of the tunica vaginalis communis. In order to comprehend its nature, it is only necessary to recall the previously mentioned bilocular hydrocele. After a direct trauma, a hemorrhagic effusion rapidly collects in the scrotal tissues, and from there extends into the cavity of the pelvis, where it is sharply defined. With this effusion as a starting-point, the one variety of bilocular hydrocele can originate by encapsulation. If of recent origin (at times increasing in size under our eyes), the tumour can not be confused with a hydrocele, as the bloody infiltration of the outer layers is characteristic. The final outcome of encapsulation is, as has just been stated, the one form of bilocular hydrocele.

In addition to the simple funicular hydrocele a *multilocular* is also found. The name merely indicates that the hydrocele of the cord, instead of being single, with smooth surface (either cylindrical, pear-shaped, or rounded in form), is composed of several knobby masses, but otherwise possesses all the attributes of an ordinary hydrocele.

There is also a *diffuse* hydrocele of the cord, by which is meant an œdema of the cellular tissues about the vas. Only one undoubted case, however, is on record. The accounts given by older authors may most reasonably be explained by assuming that they were dealing with bilocular or multilocular hydroceles.

It ought not to prove difficult to diagnose complicated conditions unless the cases are very extraordinary.

The simplest complication is a hernia. A hydrocele of the tunica exists, and above it a hernia may develop. The signs then consist of a reducible or tympanitic tumour above, or, if the hernia is adherent and contains no gas, at least an opaque tumour which gives an impulse on coughing. Below a translucent tumour, fluctuating and not affected by coughing, is found. The testicle is hidden or the testicle appears below; above it is a circumscribed, translucent, fluctuating tumour, and still farther up the hernia. This combination is formed by the funicular hydrocele complicated by a hernia. Very remarkable are the cases in which the hernial sac invaginates the hydrocele sac. This may take place either in a hydrocele of the tunica or of the cord. The hydrocele surrounds the hernia just as the tunica vaginalis surrounds the testicle. This is the *encysted hernia* of Astley Cooper. (As a rule, the hernia descends *behind* the hydrocele.)

The name of *hernial hydrocele* is applied to hernial sacs which contain a considerable quantity of fluid. The sac may fail to contain intestine; it then forms a translucent, fluctuating swelling, which can not be emptied if the neck is shut off from the general peritoneal cavity. The swelling can, in other cases, be emptied, but the fluid slowly flows back when the patient gets up and walks about. If intestine also descends, we find a hernial tumour, of which the dependent portion is translucent and fluctuating. When this happens in a child, we have a vaginal hydrocele, opening into the peritoneal cavity, through which a hernia has come down; the sac is composed of the processus vaginalis peritonæi. In an older subject, the anamnesis must show whether an exudative inflammation has not given rise to a serous effusion into the hernial sac.

To return to our first point of departure. Let us assume that the inguinal canal and the vas deferens are uninvolved, but that we are dealing with a swelling of the testicle. Such swellings are, for practical purposes, best divided into those of acute inflammatory, chronic inflammatory, and non-inflammatory origin.

ACUTE INFLAMMATION OF THE TESTICLE—acute orchitis—is marked by reddening and œdema of the skin, severe pain on pressure, also spontaneous pain, and swelling of the testicle. The most common errors in diagnosis are the result of confusing an acute epididymitis or an acute effusion into the tunica with orchitis. It is not difficult to make a correct diagnosis if the following points are kept in mind: The inflamed epididymis forms a hard, painful swelling, which is concave in front, and attains about the size of a thumb. The testicle rests in its concavity. We therefore find a round, soft, and slightly less tender mass, which corresponds in size to the normal testicle, embedded in the tense tumour formed by the inflamed epididymis. This mass is the testicle proper.

In acute hydrocele of the tunica distinct fluctuation can be obtained around the testicle. It is characteristic that if the testicle is fixed by skilful manipulation, its outline can be plainly felt through the surrounding layer of fluid. If the stringlike structure of the epididymis can be made out posteriorly, the inflammation is confined to the tunica. More often the epididymis is also involved, in which case fluctuation is felt only in front and somewhat laterally.

Simple orchitis is uncommon. It is characterized by the absence of all signs of epididymitis and acute hydrocele. Fluctuation is wanting in front, and posteriorly the normal or sometimes harder epididymis can be felt. Frequently all three conditions coexist, orchitis being followed by epididymitis, and these further complicated by an effusion into the tunica. The orchitis may be diagnosed by the very severe pains, both spontaneous and on pressure, by the swelling of the whole cord upward as far as it can be palpated, and by the violent pain in the loins. Usually the inflamed organ itself feels enlarged.

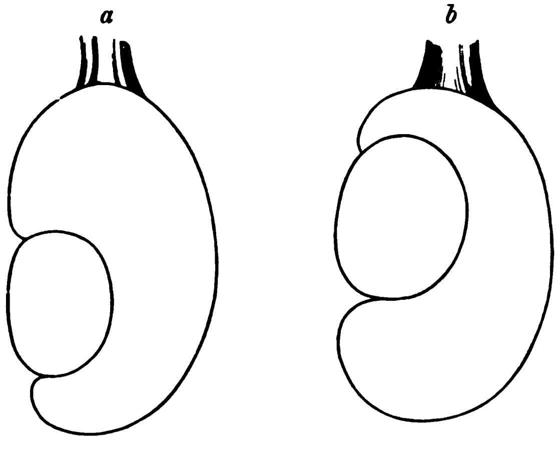

FIG. 34.—Diagram representing the conditions in epididymitis. In *a* the head, in *b* the tail, of the epididymis is more enlarged.

Formerly, it was accepted that orchitis could not occur without the most violent pain, and that the swelling of the testicle could never attain an appreciable size, because that organ is inclosed by its firm albuginea. Consequently, it was taken for granted that a severe inflammation had only one of two outcomes—either suppuration or gangrene. This is not true. The testicle can swell to twice its original size in the course of a few days, and the swelling then subside. Such cases are seen during epidemics of mumps. The epididymis increases in length, corresponding with the increase in size of the testicle.

Marked difficulty may be experienced in the diagnosis of an orchitis or epididymitis of an undescended testicle lying within the inguinal canal. Symptoms resembling strangulation of a hernia may arise, as has been previously stated. We now arrive at the discussion of CHRONIC INFLAMMATIONS of the testicle and its adnexa.

A chronic inflammation of the testicle may exist for years and produce a *diffuse* enlargement of the organ, or a chronic abscess may develop at some *circumscribed* spot. No etiological factor can be found in most cases, and an acute stage is usually wanting. Inflammatory symptoms are commonly but slightly marked; therefore it becomes evident why such cases lead us to think of a new growth, or of a hydrocele with enormously thickened walls, especially when the diffuse form confronts us. In doubtful cases, the following differential points will be of service: A hydrocele, which can be confused with a swelling of the parenchyma of the testicle, always has markedly hard spots in its sac—spots which are harder than those produced by a chronic orchitis. In orchitis we find areas of softening and harder areas, but never such stony, hard plates as in thickened hydrocele walls. In orchitis, the epididymis can usually be felt; in hydrocele, it is obscured quite early. No characteristic points of difference between a chronic orchitis and a neoplasm can be given unless we specify some exact variety of new growth. A rapidly growing neoplasm—i. e., one which forms in the course of a few months—is suspicious of a malignant tumour. In this case, the epididymis, vas, and lymph glands may already show involvement. It should be kept in mind that the retroperitoneal glands in the neighbourhood of the kidney, and not the inguinal glands, increase in size in tumours of the testicle. Tumours of slower growth can only be adenomata or cystomata. Aside from the special symptoms, which in a given case may be very striking, sudden marked

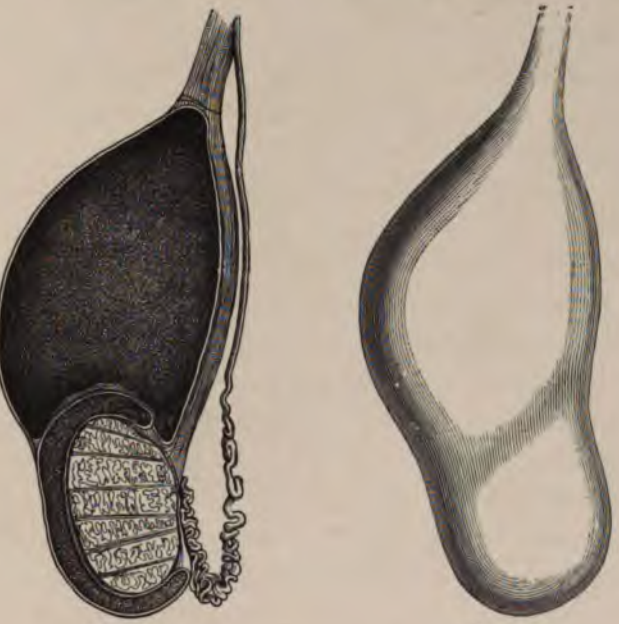

Fig. 35. Fig. 36.
Extravaginal spermatocele (diagrammatic).

increase in the rapidity of growth speaks greatly in favour of a neoplasm. Tumours, as a rule, are heavy; non-malignant tumours do not involve the skin, etc.

Chronic inflammatory tumours include both *cheesy (tubercular)* degeneration of the epididymis and *syphilitic* orchitis. Tubercular epididymitis can hardly be mistaken. Without cause, a considerable *nodular* swelling of the epididymis occurs, tardily breaks down at one point, and discharges a serous pus. The sinus shows no tendency to heal, and the whole process is unaccompanied by severe pain. Pressure upon the hard knots causes no pain. Later, the other epididymis, the vasa deferentia, the seminal vesicles, and the prostate show similar involvement. The more often examination per rectum in this condition is made, the more often early involvement of the seminal vesicles can be found—a hint which shows how unjustified the practice of castration is in these cases. The cases in which epididymitis appears after gonorrhœa, and later forms the starting-point of an acute miliary tuberculosis, are of special interest.

Syphilitic inflammation attacks the testicle itself. It runs a painless course, similar to that of a cheesy degeneration of the epididymis. The testicle also is painless on pressure, the areas of softening break down without causing pain, and the discharge is but scanty. Symptoms of syphilis in other parts of the body confirm the diagnosis.

NEW GROWTHS of the testicle form solid or cystic tumours; others are partly composed of fluid, partly of solid tissue.

Among the cystic tumours, the *spermatocele* deserves mention. If a hydrocele of the tunica has been diagnosed, but on aspiration a fluid which contains spermatozoa is obtained, the condition is called *hydrospermatocele.* This is explained by assuming that a spermatocele has ruptured into a hydrocele. But what is a spermatocele? It is best to accept Kocher's definition, that a spermatocele is a retention cyst which has developed somewhere between the rete of the testicle and the vas deferens. Not until of sufficient size do they assume a clinical significance. These cysts can be confused only with a hydrocele of the tunica or of the cord, as they are both fluctuating and translucent. From the former they are distinguished by the fact that in spermatocele the anterior and lateral surfaces of the testicle can be plainly felt, while a hydrocele first obscures these surfaces of the testis.

A hydrocele of the cord differs from a spermatocele in the following points: The spermatocele appears to lie between the testicle and the epididymis, so that the testicle is in front and the epididymis behind it. In these cases the former can not be isolated from the cyst, but in hydrocele the testicle can readily be separated from the sac. Pitha has called attention to the shape of the tumour: the spermatocele, having the testicle beneath, forms a pear-shaped mass, with the smaller end of the pear pointing downward. Although it must be acknowledged that a hydrocele may assume a similar form, a tumour of such configuration should always direct our suspicions to spermatocele. Before attempting any therapeutic interference the tumour should be aspirated, and the fluid obtained examined for spermatozoa.

In addition to the varieties of spermatocele illustrated by Figs. 35, 36, 39, in which the tumour has spread outside the tunica vaginalis propria, there is a variety of intravaginal spermatocele. In these the swelling—composed of a distended portion of the seminal duct—projects into the tunica vaginalis, above the testicle. The tumour, if of moderate proportions, gives the impression of a second testis placed above the true one.

The *solid* tumours of the testis have, from time immemorial, been divided into sarcocele and fungus. Sarcocele embraced all benign neoplasms; if any marked physical qualities were particularly striking, a further classification, such as chondrocele, osteocele, etc., were added. Medullary cancer of the testis (Breihode, pulpy testicle) is the name given to soft tumours which show great rapidity of growth. Their malignancy was so well known that in times gone by the proverb ran: The patient who refused operation survived the castrated one. To-day benign and malignant growths are operated upon without distinction, and from the practitioner's standpoint it is sufficient to make the diagnosis of "neoplasm." Even this is not made without difficulty in some cases. For instance, an hemorrhagic periorchitis—i. e., a strongly marked thickening of the

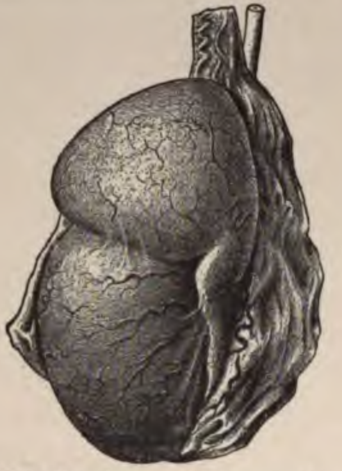

FIG. 37.—Intravaginal spermatocele.

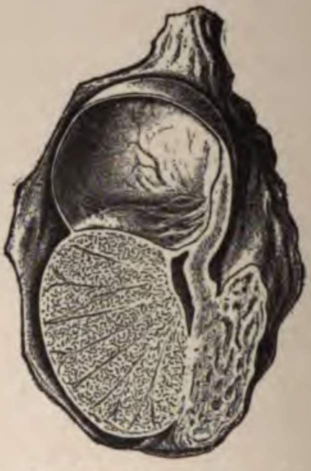

FIG. 38.—Cross-section of Fig. 37.

tunica with a moderate amount of fluid within the serous cavity—may readily be confused with a neoplasm of the testis or with syphilitic orchitis.

One of my friends, who deserves all praise both as an experienced diagnostician and as a skilful operator, had the misfortune to perform castration in a case in which he and several surgeons agreed in the diagnosis of chondroma testis. He employed Zeller's method, i. e., removed the testicle and the portion of the scrotum covering it at one stroke of the knife. The tumour proved to be nothing but a hemorrhagic hydrocele. Other operators, as Desgranges, Baum, and Kocher, found that after incision of such a hydrocele, a latent cancer (due to the inflammatory irritation), showing rapid increase of growth, unmasked itself, and eventually made castration necessary.

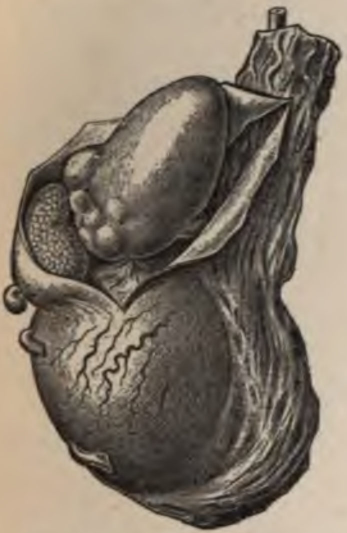

Fig. 39.—Spermatocele extravaginalis.

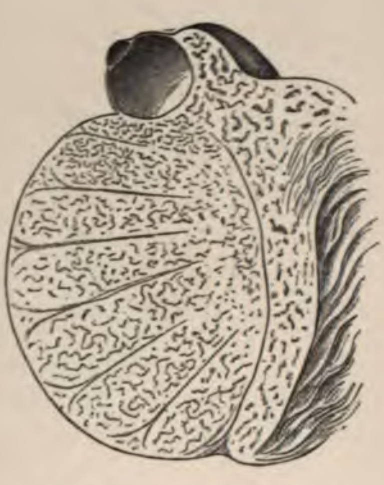

Fig. 40.—Small cyst of the testicle.

At the present day it is customary, in doubtful cases, to exclude syphilitic orchitis by antispecific treatment, unless the tumour is particularly hard. If its consistency is harder, and it has been decided to perform castration, the method of Zeller is not employed. On the contrary, the tumour is exposed and the diagnosis verified, according to the maxim, "Do as you would be done by."

In a typical case, the course run by a *malignant* tumour of the testicle does not differ from that of a malignant tumour of any other organ. Rapid growth, rapid extension from testicle to epididymis, or vice versa, then involvement of the vas, early infiltration of the retroperitoneal glands, adhesion, and rupture through the skin—these are the symptoms of malignancy. Retroperitoneal glands should always be sought for, as they enlarge early in the disease.

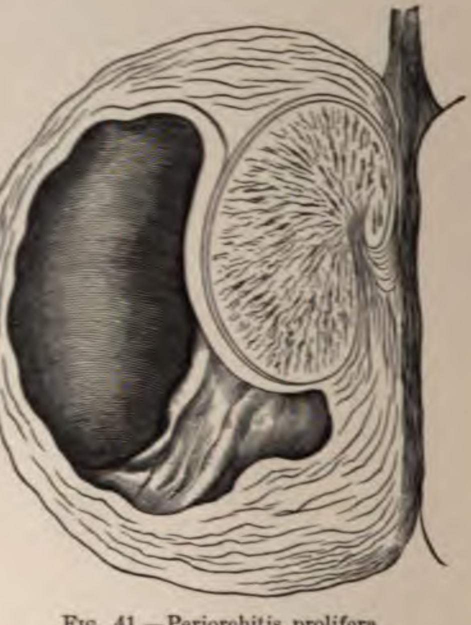

FIG. 41.—Periorchitis prolifera.

Tumours which are hard in one spot, fluctuate in another, of rapid growth, but showing no tendency to involve the epididymis and vas, and unaccompanied by enlargement of the lymph glands, may be called *cystoid.* The prognosis, however, should not be made absolutely favourable.

A congenital tumour which shows both hard and fluctuating spots is undoubtedly a *teratoma*; that is, a neoplasm which is composed of tissues which normally occur elsewhere in the body, such as skin, mucous membranes, muscle, nerves, and even bone with periosteum and marrow. The fluctuating spots may contain fluid of varied nature, mucus, serum, or sebaceous material.

Solid teratomata also occur. The tumour may be designated as a teratoma (1) if it is congenital; (2) if the testicle can be felt alongside it—for teratomata develop next to the testis; (3) if the tumour bursts open and discharges hair, teeth, or bones.

Œdema, elephantiasis, and cancer of the scrotum require no discussion from the point of view of diagnosis.

CHAPTER XXVI

DISTURBANCES OF MICTURITION; DISEASES OF THE BLADDER AND URETHRA

In the majority of cases of diseases of the bladder and urethra, the patients employ the expression "urinary trouble," which is as vague as "headache" or the popular "stomach-ache." A great number of diseases are included in these terms, and the symptoms of urinary trouble are quite as manifold. It is frequently not feasible, especially during office hours, to expose the patient immediately in order to make a local examination. Even if this does not apply, the time during which the patient is undressing may be profitably employed by putting suitable questions. This examination should consider the following points: The urinary trouble may be due to the fact that the patient can not hold the urine (incontinentia urinæ; enuresis), or is unable to pass it (retentio urinæ; ischuria), or can pass the urine only in conjunction with various abnormal symptoms (dysuria). It is evident that diseases of entirely different nature may give rise to these main symptoms.

Incontinence may be the result of paresis of the vesical sphincter, of vesical fistula, of a stone projecting into the neck of the bladder, of connective-tissue degeneration of the sphincter vesicæ, etc. *Retentio urinæ* may be due to vesical paralysis, cramp of the sphincter vesicæ, stricture of the urethra, foreign body in the urethra, urethral compression from without by a tumour or spicule of bone, displaced uterus, etc. If called to an unconscious patient, who has fallen from a scaffold and is suffering from retention, we reason differently than when a patient comes to our office with the demand that his disease should be kept secret from his friends.

The symptoms of *Dysuria* vary greatly. A careful catechism is required in order to gain exact information. A certain amount of order and method will be obtained if the following points are considered in putting the questions:

1. Is the condition of recent origin or has it existed some time?
2. Has the patient had fever or not? These two questions narrow the countless possibilities to smaller groups.
3. Has the patient passed more than the normal amount of urine within a specified time? The daily quantity of urine voided varies greatly, even among healthy people. The question is, therefore, directed to ascertain whether the patient has noticed that since a given period he empties his bladder more frequently or that his bladder shows a decreased capacity.
4. What painful sensations are felt during urination? Does vesical tenesmus exist? Is pain felt in the urethra during micturition? Is the pain more marked toward the end of the act?
5. What is the character of the stream? Has it projectile force, is the flow weak, or does the urine dribble away? Particularly find out whether the stream is winding or forked, and how long a time each urination occupies.

6. Especially important: Do the symptoms vary during a single urination, so that the stream is thin for a few moments, then thick, suddenly interrupted, only to flow again with more or less obstruction? In short, whether, during one single urination, the symptoms of all the various grades of stricture occur?

7. What is the appearance of the urine—i. e., colour, clearness, consistency, odour—and, in particular, does it contain blood?

8. Are the functions of neighbouring organs not disturbed? Do constipation and, in women, menstrual disturbances coexist?

If these points have been determined, in some cases the diagnosis can be made merely from the combination of symptoms obtained.

For instance, a patient informs us that he has during the last year occasionally passed bloody urine. During a single urination he at one moment passes water without trouble, the stream then becomes thin, then stops entirely. If he should add that change of position sometimes removes the obstruction, we could hardly draw any conclusion but that a foreign body in the bladder, a vesical calculus, caused obstruction by assuming a position over the internal urethral orifice. By changing its site it would bring about various degrees of obstruction during the course of a single act of micturition. Naturally, a pedunculated growth in the bladder could cause the same symptoms, but such growths are extremely rare. Sounding of the bladder, chemical and microscopical examination of the urine, must then be employed to arrive at a positive conclusion.

In another case, the patient tells us that every fifteen, or even every five, minutes he has an overwhelming desire to pass water—so strong that he has to jump out of bed. He passes a few teaspoonfuls, or only a few drops, of urine, and accomplishes this at the cost of much fear, a drenching sweat, and by aid of peculiar manipulations and positions. The trouble then can be due only to *Cystitis* or *Concentric hypertrophy of the bladder* following a stricture. In order to distinguish between these two diseases, it is only necessary to find out whether the condition began suddenly or was of slow and gradual development. Chronic hypertrophy is the result of a long-standing tight stricture. The patient dwells upon single epochs of his trouble, so that the gradual increase of his symptoms can be explained in no other way than by a cause which has a slow but constant action. Cystitis sets in suddenly, the accompanying symptoms are of greater or lesser intensity—fever, occasionally vomiting, and in some cases typhoid prostration mark its onset; in the latter class of cases the patient does not come to our office. In milder cases the patient, after the first severity of the onset has passed, never fails to harp on the sudden beginning of vesical tenesmus and chills, on the wretched night he passed, although quite well on the day before. It may, however, happen that a chronic cystitis of long standing develops a sudden acute exacerbation. This gives the picture of a tedious illness with manifold symptoms, and finally the onset of uncontrollable tenesmus, just as in concentric hypertrophy of the bladder. But in cystitis the urine is hot and irritating, cloudy, alkaline, and sometimes bloody.

To multiply these instances, let us consider a woman toward the end of her teens. Dysmenorrhœa and sterility lead us to suspect anteflexion; backache, metrorrhagia, constipation, all point to retroflexion.

All in all, we may say that the diagnosis is narrowed down to a few conditions which resemble each other, provided the patient's surroundings, age and sex, and the duration of the disease are taken into account. Percussion of the hypogastrium, external palpation of the urethra, rectal examination of prostate and bladder in the male, vaginal examination in the female, must be resorted to. Chemical and microscopical examination of the urine and catheterization help to confirm the tentative diagnosis yielded by the history.

These general introductory remarks will now be followed by a more detailed account of the various diseases arranged in groups.

The first group comprises INJURIES to the urethra and bladder.

Subcutaneous injuries of the urethra are due either to *crushing* or *tearing* violence. A tear may be suspected if, in addition to the symptoms of trauma—ecchymosis and pain along the course of the urethra—blood flows from the meatus. Bruising without rupture may be diagnosed by the absence of bleeding from the urethra, although the other symptoms of trauma are present, and urination causes a feeling of pain and obstruction at the injured spot.

A subcutaneous *rupture of the bladder* is the result of severe trauma to the distended viscus. The patient vainly tries to satisfy his constant desire to urinate; the physician is unable to draw off a sufficient amount of urine; at the most, a small quantity of bloody fluid is obtained. If the extravasation is extraperitoneal, the symptoms of urinary infiltration develop; if the bladder with its peritoneal coat has been torn, peritonitis sets in.

Cases of dysuria or retention met with after fractures of the pelvis frequently remain unexplained. They occur without injury to the bladder or compression of the urethra, which might account for the symptoms.

Constant flow of urine through an *open* wound, either out of the rectum or from a skin wound, can be explained only by assuming an injury to the bladder; leakage of urine confined to the time of micturition is characteristic of an injured urethra. Probing the wound or examining it with the finger while a metal catheter is introduced into the urethra confirms these facts. This is especially necessary if the injured individual has previously suffered from incontinence. In this case, a urethral wound might cause *constant* dribbling.

We must not fail to warn the beginner that he should never forget to catheterize patients suffering from injuries to the head or spine, or from concussion of the brain. It is pardonable if the beginner's attention is at first directed to the severe local injury, which may cause him much anxiety; but it is unpardonable if he forgets to empty the bladder, or at least to inquire about the patient's urination.

A second group may be formed by contrasting conditions accompanied by ACUTE ONSET of *strangury, with or without retention.*

If the patient has fever, and the main symptom is strangury with the discharge of a few drops of cloudy or bloody urine, acute cystitis, prostatitis, or abscess of the bladder wall must be thought of. All these conditions arise acutely, and may be accompanied by the most severe constitutional symptoms, such as typhoid state, prostration, vomiting, hiccough, stupor, and coma.

Prostatitis causes difficulty in defecation. The cutting pain radiates from the prostate to the symphysis and toward the loin, and is also felt along the thigh. Rectal examination—even the most careful introduction of the finger may cause intense pain—shows the prostate to be enlarged, excessively sensitive to pressure, hard, or already fluctuating. Sometimes, after withdrawing the finger, blood and pus flow from the urethra.

In *abscess* of the bladder wall a painful swelling is felt in the region of the bladder. It is dull on percussion, vaguely fluctuating, and persists after the bladder is emptied. If these symptoms are wanting, acute *cystitis* is diagnosed by exclusion.

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Other causes of strangury and retention may be recognised at once. A severe gonorrhœal *urethritis* is diagnosed by the discharge. A traumatic *cavernitis* is characterized by the history of trauma, the visible swelling, the deformity of the penis. Old men should be examined for prostatic hypertrophy. Pelvic tumours which cause retention by pressure on the urethra are felt when a rectal examination is made. True retention is produced by tumours only when the distorted and displaced urethra becomes congested after some local injury.

Another group is formed by CHRONIC cases of dysuria. *Strictures of the urethra* are the chief members of this group.

In some cases the description of the symptoms obtained is not sufficiently characteristic to enable us to arrive at a diagnosis; in other cases the complaint is unmistakable. For instance, the patient relates that he had an attack of gonorrhœa some time ago—it may have been many years before. After one or more years he noticed that each act of micturition occupied a longer period of time, and that, in addition to the slower emptying of the bladder, he was often troubled by tenesmus. Finally, the stream could be started only after considerable time spent in straining. The stream was visibly altered and the urine no longer clear, a cloudy drop appearing at the meatus. Ejaculation caused pain at the base of the bladder, and the force with which the semen was ejected had greatly diminished.

This complex gives the complete picture of a slowly increasing urethral *stricture*. The obstruction to the flow of urine and to the ejaculation of semen indicates a diminution of the lumen; the cloudy drop, containing threads, shows that a catarrh has developed behind the point of stricture, and the cloudy urine is due to the chronic cystitis which accompanies the condition.

Local examination is, of course, indicated. The urethra is palpated with the thumb and index-finger as far as the membranous portion. Frequently the stricture can be felt and its length and consistence approximately estimated. By introducing a metal sound, these characteristics are more readily determined. It is advisable to use a stone-searcher of as large a calibre as the urethra will tolerate. In this manner a stricture can not be overlooked, and the spot at which the obstruction *begins*, or, in case more than one is present, the beginning of the first stricture, can be ascertained. We then introduce a smaller instrument, until we finally reach one which passes through the stricture. Instead of a searcher, it may prove of advantage to use an olive-pointed bougie. In withdrawing the bougie, the *length* of the stricture can be estimated, for during the withdrawal of the olive the instrument is clasped more firmly by the strictured portion. If the stricture is very tight, fill the urethra with filiform bougies (catgut bougies), and try each one in turn. One is sure to lie in the opening of the stricture.

Some points in the symptomatology require further detail. The site and nature of the stricture can sometimes be ascertained by the peculiar change in the character of the stream. One of the most experienced of genito-urinary surgeons, Professor Dittel, of Vienna, teaches the following: A stricture of the posterior urethra, if of large calibre, gives a rather thick stream without projectile force. If the stricture is tight, the urine appears in small, disconnected drops. Stricture of the anterior urethra allows the urine to flow with greater velocity: if the stricture is narrow and short, the stream is split; if moderately long, the urine flows in a short curve. Involuntary dribbling after micturition indicates a dilatation behind the stricture. If, in addition, the dribbling continues throughout the intervals, we must conclude that the superficial sphincter of the bladder has been incorporated in the callus, and has undergone fibrous degeneration—this occurs in extensive strictures of long standing which are palpable externally—or that it has become paralyzed, which takes place in recent stricture of small extent, situated in the penile portion.

Among the causes of chronic obstructions, *vesical calculi* occupy an important place. Patients suffering with stone state that they pass blood at varying intervals. They are troubled with a dull pain localized in the perineum, increased during exercise, such as walking or driving. Change in the character of the stream is especially characteristic. The patient starts to urinate, and the stream is strong and full for a few seconds. Suddenly, the flow is interrupted; a few drops are passed, but gradually the urine begins to flow more freely, the stream broadens, only, however, to narrow once more or stop entirely. Just as suddenly, the urine is again passed with normal force. Thus all grades of obstruction, from normal urination to complete stoppage, occur during a single emptying of the bladder. Every patient suffering with this trouble has learned by experience that certain manipulations will aid micturition, and that the obstruction may often be relieved by change of posture. If the anamnesis brings out these points, we at once turn to the stone-searcher as the most suitable instrument to employ in the local examination. We select the thickest instrument which can be passed without great discomfort. Sounding enlightens us concerning the width of the urethra, the presence or absence of strictures, possible prostatic hypertrophy, etc. Inspection of the penis may show that the prepuce has been elongated by traction made by the patient to relieve pain. Retracted scars point to healed fistulæ or strictures, due to the passage of stones at some previous date.

If the bladder is reached without hindrance, manipulation of the instrument gives certain information. The searcher is first passed backward to the posterior wall of the bladder, and then pulled forward once more. It is then turned to the right and to the left. This manœuvre not only enables us to look for a stone, but also to measure the capacity of the bladder approximately. By passing the tip along the wall, we feel trabeculæ if well developed, and in certain cases are able to form some conception of the sensitiveness of the mucous membrane.

In regard to the stone, it is desirable to determine its position, size, shape, hardness, and chemical composition. Each of these points has its own significance. It is not sufficient to discover the actual position of the calculus. It must also be determined whether the stone is freely movable or fixed in its place.

If the stone is found in the same spot at repeated examinations, if it is unaffected by the patient's change of position (while the searcher is in the bladder), or if it presents the same area whenever the sound is passed over its surface; if the hemorrhage and dysuria are insignificant, either from the start or after a certain period—it is safe to assume that the calculus lies in a diverticulum. Sometimes the stone plays hide-and-seek with the surgeon. To-day it is readily found, to-morrow it has disappeared; one physician finds it, another can not feel it, and doubts its existence. Von Dumreicher used to tell several amusing stories of this kind. He assisted Wattman at a lithotomy, in which no stone was to be seen when the bladder was opened. The bladder had partially contracted in the shape of an hour-glass, of which the upper half contained the stone, the lower being empty. Pressure above the symphysis released the stone. A similar observation was made as early as the time of P. Franco.

In order to form some conception of the *size* and shape of the stone, it is necessary to grasp its various diameters between the arms of a lithotrite. A rough estimation can be gained by rectal palpation.

The *hardness* of the stone depends upon its composition. Chemical and microscopical examination of the urine is frequently an insufficient guide. Sounding produces a note when the stone is struck, but large experience is required in order to interpret this note correctly. Examination with the lithotrite may be employed not only to determine the resistance offered to the closure of the instrument, but also to break off fragments which can be examined chemically.

The various kinds of calculi act differently to the pressure exerted by the lithotrite.

When a *phosphatic* calculus is grasped, it gives the impression of a compressible, sandlike body.

A *uric acid* calculus offers a firm resistance. If the screw is tightened, the outer layers are crushed, and consequently the branches of the lithotrite approximate. When the pressure is suddenly released, the screw does not recoil.

The resistance offered by a stone composed of *oxalates* is very great. This variety of calculus does not seem to be compressed or crushed when the lithotrite blades are tightened; moreover, when the pressure is suddenly released, the blades snap back.

Cystin calculi are of the consistency of wax.

This instrumental examination is of great importance in supplementing urinary examination, which can only show the composition of the outer layers of the stone.

Chronic obstruction may be due to *disturbances of innervation* of the bladder. In these cases, great difficulty is often experienced in emptying the bladder.

The first drops are passed only after severe straining; the duration of the whole act is greatly prolonged; the stream is thin and weak, and yet no mechanical obstruction can be found. If the patient is advanced in years, we suspect hypertrophied prostate. Sounding shows the lumen of the urethra to be unobstructed, the bladder free from foreign bodies; rectal examination shows a prostate not especially enlarged. The difficulty evidently lies in a paralysis of the *detrusor.* This can be demonstrated in the following way: The patient is asked to empty his bladder, and after he has finished passing his urine he is catheterized. Several ounces of residual urine are withdrawn by the catheter.

Spasmodic contraction of the sphincter vesicæ may produce symptoms of dysuria of marked intensity—*spasmodic stricture.*

In an extremely interesting case, observed by v. Dumreicher, the symptoms corresponded exactly to those seen in concentric hypertrophy of the bladder. The sound detected no stricture, the urine was normal; the whole disease evidently was a neurosis. The patient, who had formerly been obliged to urinate every ten minutes, was cured by an artifice practised by v. Dumreicher. He engaged the patient in a conversation lasting two hours, thus conclusively proving to him that he could hold his urine. Dittel records another case: A patient, about to go on a journey, was suddenly attacked with retention, and the physician who first treated him found an obstruction. Dittel was confronted by the same obstruction, but when he left the catheter in the urethra for fifteen minutes the instrument suddenly entered the bladder. Von Dumreicher encountered a similar case, the patient being a physician. A sixth of a grain of morphine sufficed to relieve the spasm. Psychical causes may produce spasm of the bladder or urethra. Gouthrie relates that a certain lawyer was invariably attacked by retention before he argued an important case; similarly a clergyman, who was affected before he preached. Some people suffer with this trouble—strangury, so-called—after drinking freshly brewed beer, others after coitus, still others after coitus if they have previously partaken of fresh beer. Patients suffering from pyelitis, arthritis, or diabetes may be subject to spasmodic stricture; fissures of the anus, worms, etc., may produce it by reflex irritation.

We have previously mentioned that the symptoms produced by a stone are also caused by a pedunculated tumour within the bladder. The diagnosis can only be made with the searcher, and repeated examinations may be necessary if the neoplasm is covered with incrustations.

Carcinoma of the bladder, which most frequently is of the villous variety, produces a different picture. The symptoms are those of a chronic cystitis. A positive diagnosis can be made only after discharged portions of the tumour have been examined under the microscope. A tentative diagnosis can be made if the patient is well advanced in years, if the cystitis increases in intensity in spite of treatment, if frequent hemorrhages occur. Additional evidence is furnished if rectal examination reveals a painless resistance, if carcinoma of other organs can be demonstrated (especially in the neighbouring lymphatic glands), and if cachexia is an early symptom.

Papillomata of the bladder, sometimes multiple, produce severe hemorrhages, but are unaccompanied by the other symptoms of malignancy which are present in cancer. They develop slowly; the hemorrhages recur at irregular intervals, and the whole course extends over many years.

Urinary disturbances which produce symptoms that do not agree with any typical disease must lead to the suspicion of a foreign body. This does not mean, however, that foreign bodies in the bladder may not give the typical symptoms of vesical calculus. At times it is impossible to recognise the nature of the incrusted mass before it has been extracted. In other cases, a body may remain for years in the vagina and then suddenly produce the most manifold disturbances of micturition, so that the whole picture may be very complicated. In every case, in man or woman, in young or old, the oddest articles may be found by digital exploration or sounding. The presence of broken ends of bougies or catheters are most readily explained. In the male urethra, pencils, paint-brushes, brush handles, straws, thin straps, etc., may be introduced by masturbators. Women may introduce needles, needle-cases, and similar articles for the same purpose. Pebbles, marbles, seeds, etc., are occasionally found. Dieffenbach relates that he removed a fork, five inches in length, from a man's bladder, by perineal section.

CHAPTER XXVII

CONDITIONS MET WITH IN VARIOUS DISEASES OF THE BLADDER

STRICTURE, prostatic hypertrophy, lithiasis, and foreign bodies in the bladder are frequently accompanied by cystitis. Hæmaturia is often met with in vesical calculus, neoplasms, and many other conditions. Any interference with the bladder, litholapaxy, suprapubic or perineal section, even catheterization, may be followed by so-called urethral fever. Injuries, ulcerative perforation of the bladder or urethra, may produce urinary infiltration. Not infrequently uræmic intoxication of the blood confronts the surgeon. All these conditions occur in the course of various diseases, and will therefore be briefly discussed in this chapter.

The gross pathological changes of the urine demand our first attention.

The threads appearing in the urine in cases of stricture point to a catarrh of the urethral follicles, but they also occur in prostatitis or in hypertrophy of the bladder. In order to determine their origin, it is necessary to observe whether they appear at the beginning or end of urination. The patient is directed to urinate into three glasses. The first of the urine is collected in one, the chief quantity in the second, and that portion which is finally squeezed out of the bladder in the last.

The CHRONIC CYSTITIS which accompanies stricture may vary in intensity. Pyelitis and nephritis may have already developed.

Chronic cystitis may conveniently be divided into three degrees. In the mildest grade, the urine is slightly turbid, and a *cloudy sediment* deposits on prolonged standing. An alkaline reaction rapidly sets in—that is, in the course of one to two hours—or the urine may show mild alkalinity from the outset. The chemical composition is normal; the sediment is composed of ammonio-magnesic phosphate, bladder epithelium, and so-called mucous cells (Schleimzellen). The second grade is characterized by the occurrence of *pus*. The pus is recognised macroscopically by the viscid sediment which adheres to the tube. This quality is due to the action of the ammonium carbonate on the pus. The urine is alkaline from the start, cloudy, and of ammoniacal odour. The most serious form is characterized by *stinking* urine. The strongly alkaline urine is dirty brown or greenish. Its odour is putrid; a strong odour of sulphuretted hydrogen often predominates. The urine may contain blood pigment.

PYELITIS may also be divided into three degrees of intensity. The mildest grade is characterized by normal amount of urine. The urine is cloudy, *acid* in reaction, contains traces of albumen and a few pus cells. A more severe involvement of the pelvis, *suppurative* pyelitis, produces a pale, cloudy urine, with a plainly visible sediment, the reaction still remaining *acid*. The sediment is never viscid; it consists of pus, of which each cell is plainly distinguishable under the microscope. The amount of albumen corresponds to the amount of pus found in the sediment. Pyelitis of the third degree is characterized by a complicating parenchymatous affection of the kidneys. The albumen is more abundant than can be accounted for by the quantity of the pus, and the amount of urea excreted during the twenty-four hours is lower than normal.

Cases in which blood is contained in the urine—HÆMATURIA—are more difficult to recognise.

Bleeding from the kidney is recognisable in the urine only if the hemorrhage has taken place in or distal to the tubuli uriniferi. As the blood coagulates in the tubules, fibrin casts coated with red blood-cells are found microscopically, and may be regarded as characteristic of renal hemorrhages. Any one who has made a practice of examining the urine of such cases will recognise bleeding from the kidney by the appearance of the urine, which has a dirty brown-yellow colour. Bloody coagula, several inches in length, resembling the ordinary round-worm, if found in the urine, are very suggestive. They are casts of the ureter, and usually cause colicky pains during their expulsion. The chemical examination shows the chief symptoms of kidney disease—acid urine, more albumen than is accounted for by the amount of blood present, a low specific gravity, diminution of urea, calcium and other phosphates, etc.

Hemorrhage from the bladder occurs only in vesical affections, consequently the urine shows the signs of a chronic cystitis, or the symptoms complained of by the patient point to some of the diseases of the bladder which have been discussed above. The urine, therefore, is found to be strongly alkaline, and contains ammonium carbonate, with the viscid sediment with which we are familiar. No casts are seen; the clots are rounded with irregular outline, and inclose phosphates. Microscopically, triple phosphate and bladder epithelium appear in great quantities. Chemically, we find the specific gravity within normal limits, and the urea, in the twenty-four-hour specimen, is not diminished.

To determine whether the blood comes from the bladder or from the kidney is, at times, one of the most difficult problems in diagnosis. And even if we take for granted that the hemorrhage is from the kidney, it frequently is impossible to fathom the cause of the trouble. Neoplasms, calculi, and tuberculosis are the more frequent causes. At times, after we have been compelled to resort to a nephrotomy, it is found, at the operation, that the kidney is not diseased. This condition, called by the French *hémophilie rénale,* is very mysterious.

Bleeding from the bladder, occurring in middle-aged men afflicted with prostatic hypertrophy, may be solely due to this affection. Such hemorrhage is most apt to occur after physical exertion.

When unmixed blood flows from the urethra it may be accepted as a sure sign of *urethral hemorrhage.* If the patient urinates, the blood and urine appear unmixed, or the urine contains small, fresh clots. That the causes may be manifold is readily realized from what has been previously said.

My friend, Professor Mosetig, in Vienna, surprised me by demonstrating the etiological cause in a case of bleeding from the urethra. None of the ordinary causes could be found. Mosetig turned to the man and asked him whether he had not tied a string around his penis. The patient at once confessed.

URETHRAL FEVER is the English appellation for what in Germany is known as *Reaction* of the urinary tract. What does this mean?

It is well known that nervous subjects may after catheterization have a chill, which is not followed by further consequences. In an hour or two the patient is quite well, and remains so. As no other explanation than a reflex nervous disturbance can be offered, the name of *nervous* reaction has been applied. In distinction to this, Dittel places the so-called *morbid* reaction.

Patients suffering from kidney or bladder disease may, after attempts at catheterization, dilatation of a stricture, or sounding of the bladder, have a chill within the next twenty-four hours, and, *in addition*, an *exacerbation* of the pre-existing affection of kidney or bladder. This is followed by a protracted illness, characterized by delirium, gastro-enteritis, and typhoid condition. If the *nervous* reaction is of violent character, subsequent to the chill, protracted illness may develop, but the urinary organs remain undiseased—i. e., in statu quo. To distinguish a nervous from a morbid reaction we must take into consideration the character of the urine, both chemical and microscopical, diminution in the daily quantity excreted, etc.

According to Dittel, we need not wait for the report of the urine, for persistent headache and absence of complete remissions point to a morbid process. If the urethral chill occurs after *injury* to the urethra, such as may be caused by attempts at dilatation, the reaction is called *traumatic*.

As a rule, if normal acid urine is injected into the tissues of an animal, no local reaction occurs unless the technic is faulty. After operations performed upon the human subject, if acid urine flows over the fresh wound surface, reaction does not usually take place. This observation influenced Simon, and later Menzel, to experiment upon the effect of urine on healthy tissues. They proved that alkaline urine kills the tissues and causes sloughing. Acid urine, however, can also prove fatal to the tissues under certain conditions; this is due to the influence of bacterial action.

In one of my wards a careless orderly perforated the rectum while giving an enema, and the fluid (oil and lukewarm water) was injected into the perirectal tissues. The resulting picture was an exact counterpart of urinary infiltration. The tissues sloughed and the process spread along the perineum, scrotum, and inguinal region. As I was not informed of the accident, I first noticed the local symptoms in the perineum and diagnosed urinary infiltration. But the genito-urinary tract proved healthy, the urine normal. After the true cause had been acknowledged, I found a hole in the rectum at the site of a varicose ulcer. Autopsy showed that the urinary organs were normal. Bacterial infection here, too, was the main factor.

Whatever the cause of URINARY INFILTRATION, the symptoms are those of a deep sloughing process, analogous to a sloughing wound, which spreads to the neighbouring tissues, infiltrates and destroys them. The appearance and course of such an infiltration—assuming that it occurs in the perineum—is as follows: The perineum is swollen, dark red, hot, and painful; its margins are edematous. This change spreads rapidly to the scrotum and penis or mons, extends upward onto the belly and downward to the groin. By the time that new regions are involved, the original focus is covered with blebs. These vesicles contain darkish fluid, and scattered about are dark-blue to green spots, with irregular dentate margin. The tissues feel hollow; an emphysematous crackling can be felt, and if early incision is not resorted to the whole area breaks down into a fetid slough. The patient suffers from general symptoms also, a chill at the onset, and fever during the course of the disease.

The point at which the infiltration takes place varies greatly, and may be situated in any portion of the urinary tract from the pelvis of the kidney to the external meatus of the urethra. I saw a case of urinary infiltration result from a point outside the urinary passages. The trouble was due to an ulcer on the inner surface of the prepuce, causing phimosis. The condition was at once recognised after the prepuce had been split. The course taken by the extravasated urine depends upon the relation of the site of rupture to the triangular ligament—i. e., the deep layer of the perineal fascia. The rupture may be anterior or posterior to this structure. If the suprapubic region and the tissues above Poupart's grow red and edematous during the course of a pericystitis in the space of Retzius, the rupture usually is situated behind the triangular ligament. On the other hand, similar symptoms first appearing in the scrotum and perineum point to a rupture anterior to the membranous urethra. It is not always possible to pass a sound in these cases.

The patient is frequently able to inform us of the exact time of rupture. At the time of the accident he noticed a desire to urinate, followed by a deep, tearing pain. Although only a little urine and blood was passed, the bladder felt completely empty.

URÆMIC INTOXICATION is a symptom-complex which results from advanced kidney disease or from some mechanical obstruction to the excretion of the urine. The true cause of uræmia has not yet been determined. The symptoms may appear either rapidly or slowly. As a rule the first symptom is headache, followed by somnolence or apathy, which may reach an extreme degree. Vomiting, often of most persistent and uncontrollable character, is added to these, or convulsive attacks, most commonly epileptiform, rarely tetanic in nature, take its place. The patient lies completely comatose, his respiration grows stertorous, the convulsions recur again and again, and death from paralysis of all the functions ends the scene. Uræmia may run a more chronic course. The most severe attack may pass off and the patient regain a comparative degree of health, but the uræmic condition recurs in the course of weeks or months.

Such attacks appearing during the course of a severe kidney disease may well raise the question as to whether the cause is uræmic, or whether the condition is due to an œdema of the brain. I shall not dilate on this theme. I desire to cite one more special case. A patient who has repeatedly suffered with renal colic, and perhaps even passed renal calculi, is now subject to complete suppression. We know that he has no advanced kidney trouble. Examination shows that the bladder is empty. The bladder is again found empty after several hours have been allowed to pass. If the anuria continues for several days, the condition, usually ascribed to uræmia, is probably due to a horseshoe kidney, whose single ureter has been blocked by a calculus.

Finally, URINARY FISTULÆ, which are found as complications of many previously existing conditions, require mention.

Urinary fistulæ in the male may be situated in any part of the urinary tract; their cause, as a rule, is stricture. Internally, they may open into the urethra or bladder; externally, on the scrotum, penis, perineum, the fold of the groin, on the pubes, the hypochondrium, the upper part of the thigh, or into the rectum.

I had a patient who, as the result of stricture, had two fistulæ which opened into the rectum, several which opened on the scrotum, perineum, thigh, and one in the gluteal region. Just beneath the navel a painful abscess developed, which was at once recognised as a beginning fistula. Strange to say, the patient did not seek hospital treatment until this abscess formed.

At times difficulties in diagnosis may arise in connection with these fistulæ. The first point to determine is, whether the fistulous tract leads down to the urethra or not. Sometimes the drops of fluid which trickle away can not be distinctly proved to be urine. A metal sound is passed along the urethra, a probe into the fistula, and it is then determined whether the instruments come into contact. If the course of the fistulous tract is sinuous, these means may fail. It may be necessary to inject coloured fluids into the urinary passages in order to see whether they appear through the fistula. In the next place, it may be necessary to determine whether the internal opening lies in the urethra or in the bladder. If the urine appears only when the patient urinates, the opening is in the urethra. The urine may dribble continuously, and yet the opening may be in the urethra, provided the patient is subject to incontinence from some other cause. This point has already been spoken of in connection with injuries. It should again be emphasized here, because incontinence is more often found as the result of urinary fistula than of trauma. Careful sounding may decide. Dittel very properly remarks that the spot at which the sound and probe *first* come in contact is the internal opening (they may come in contact inside the bladder and thus give a false impression).

Urinary fistulæ in the female may open in various places. A ureteral fistula may have its outlet on the belly wall. The first nephrectomy performed by Simon was in just such a case, the fistula dating from an operation previously performed. In the majority of instances the fistula opens into the vagina, and most commonly is due to parturition. The diagnosis in these cases must be directed to determine two points: 1. Does a fistula exist? 2. Of what variety is it?

The first point requires careful examination only in case the fistula is very small. In fistulæ of larger size, introduction of the finger into the vagina at once detects the defect, and if the opening permits, the finger may be passed into the bladder. This examination will also allow us to recognise the shape of the opening, the thickness of the edges, unevenness of outline, etc. If the fistula is minute, careful examination of the patient, placed upon the gynecological table, is required to discover its existence. Sometimes coloured fluids, such as milk or India ink, must be injected into the bladder in order to demonstrate the fistula by watching for the spot in the vagina in which the fluid appears.

In regard to the second point. We distinguish: 1. Urethrovaginal fistulæ, which allow the urine to escape only during the act of micturition; therefore, no incontinence. 2. Vesicovaginal fistulæ, through which the urine constantly escapes. 3. Ureterovaginal fistulæ, which are characterized by a combination of these symptoms, for here the dribbling is constant, although periodical voluntary emptying of the bladder also occurs. The sound ureter empties into the bladder, and gradually fills it. The diseased ureter empties into the vagina, and causes the dribbling. A ureteral catheter, passed into the fistula, enters the ureter, but not the bladder. Coloured fluid injected into the bladder naturally does not escape per vaginam. We assume that that portion of the ureter which is between the bladder and the fistula has been obliterated, but even without this, fluid injected into the bladder would but rarely penetrate into the ureter. By introducing a bent probe through the fistula, efforts can be made to reach the bladder through the ureter, and there bring the probe in contact with a metal sound previously introduced through the urethra.

The fistula may be so situated that it opens at the internal urethral meatus, and thus strikes both urethra and bladder. This condition is called a urethro-vesicovaginal fistula. A vesicovaginal fistula may have its opening placed in such a fashion that part of the circumference is formed by the uninjured cervix—a superficial cervical fistula—or part of the cervix may have been sacrificed and the rest form the edge of the opening—deep vesicocervical fistula. Finally, the opening may be situated within the cervical canal; this is called a vesicouterine fistula. The urine or fluid injected into the bladder appears at the external os.

A ureteral fistula opening into the cervix (uretero-uterine) will be characterized by periodic emptying of the bladder, and continues dribbling of urine from the cervix. Fluid injected into the bladder does not appear at the os. If the cervix is dilated by sponge-tents, the fistulous opening may be seen and probed.

If both ureteral and vesicovaginal fistulæ exist in the same case, the symptom of periodical micturition is wanting. Here accurate and careful probing is necessary. The instrument introduced into the bladder never comes in contact with the probe introduced into the ureter, and this last-mentioned sound passes far up into the region of the kidney.

Kinking or spiral course of the ureter requires a flexible catheter. In such cases other phenomena occur, such as cessation of flow from the ureter for a longer or shorter period, with pain in the loin due to the impeded outflow of urine. This is followed by the sudden discharge of larger quantities of urine, with immediate relief from the pain.

In conclusion, a condition which may occur in the course of various diseases of the genito-urinary tract, *anuria*, may be mentioned. By this is meant the cessation of the flow of urine into the bladder. If a patient has bilateral renal calculi, it might happen that both ureters became obstructed at the same time. No urine would then enter the bladder, and bilateral hydronephrosis would be the outcome. If the patient has only one ureter (*one kidney*) this accident might occur more readily. Under anuria we include the conditions in which the kidneys do not secrete urine. If this occurs in severe renal disease, its cause is, of course, readily understood. Such cessation of secretion may also, however, be due to purely reflex irritation.

CHAPTER XXVIII

DISEASES OF THE PENIS AND VULVA

DISEASES of the penis require but little discussion. Benign neoplasms are of very rare occurrence, if we except the *gonorrhœal condyloma* which is frequently met with. Epithelioma of the penis should offer no difficulty in diagnosis, if the age of the patient, and the marked induration of the ulcer, both at its base and edges, are taken into account. The evident involvement of the surrounding tissues by extension—in marked contrast to the destructive progression of an ulcer—the shotty inguinal glands, and the duration of the trouble must also be considered. Sometimes epithelioma of the penis is found in the form of a nodule within one or both of the corpora cavernosa. It is at once recognised by its hard consistency. Frequently a primary cancerous growth of the glans is followed by metastatic nodules in the corpora cavernosa.

In cases of paraphimosis, remember to look for a thread tied about the penis, as cause of the trouble. Venereal processes will not be considered.

CAVERNITIS may result from trauma or from suppuration in the neighbourhood, followed by perforation into the corpora. It occurs, therefore, both in the form of a local, circumscribed, or a general inflammation.

The most striking symptom is the distortion of the penis, for, as a result of the excessive infiltration of a portion of one corpus cavernosum, the penis is bent toward the opposite side. If cicatrization then follows, the organ is bent in the reverse direction—i. e., toward the diseased side—by the cicatricial contracture. The most unpleasant after-effect is the resulting deformity of the penis during erection. If the cavernitis was bilateral and cicatrices form on both sides, the organ becomes erect as far as the scar tissue, beyond this it remains flaccid.

An acute cavernitis of considerable extent, with rapid pus formation, produces an alarming picture. The penis is enormously swollen as far as the perineum, and the skin of the penis becomes markedly edematous. A sharp line of demarcation—just as if part of the swelling had been cut off—is characteristic. The disease occasionally is seen during the course of a gonorrhœa which has been subjected to too energetic treatment. After fluctuation appears, incision is followed by the evacuation of large quantities of pus.

Functional disturbances, especially IMPOTENCE, require special mention. A common variety is due to *psychical* causes. To explain this phenomenon, an inhibitory centre, situated in the lumbar cord, has been assumed. This centre can be stimulated by the imagination, so that erection grows impossible in spite of the extreme desire of the individual. Not later than in the last century such cases of psychical impotence were judged to be the result of witchcraft.

There are other forms due to actual pathological changes. The physician must regard the matter in a very serious light when he is taken into the patient's confidence, for the most varied psychical disturbances, and even suicide, have resulted from impotence. Sometimes the true condition of affairs is concealed from the physician, and patients afflicted with impotence will wander from doctor to doctor, complaining of various troubles, until they finally come to a physician who asks if they are potent. This physician at once gains their confidence. As most men are ashamed of their disease, the question must be put categorically.

A man who is able to have coitus and to ejaculate may yet be impotent if unable to inseminate. If the ejaculated fluid fails to contain spermatozoa, the condition is denominated *Azoospermia*. The cause must be an atrophy of the testicle, especially atrophy or obliteration of the lumen of any of the efferent passages, such as the vas or epididymis, following an attack of gonorrhœa. This condition should always be looked for. Another form of impotence is known as *Aspermatism*. Erection is normal and the patient can have connection, but no ejaculation is possible. If the patient never ejaculates semen, the condition is called *permanent* aspermia (so named by B. Schulz). If semen fails to be voided only during coitus, but pollutions occur, the condition is known as *temporary* aspermia. The latter is of lesser significance than the former, for it is purely the result of abnormal nerve control. Permanent aspermia is due to closure of the ejaculatory ducts, unless it is caused by stricture of the urethra. If the ejaculatory ducts are strictured, no spermatozoa can be found in the urine, but if the openings of the ducts are displaced, spermatozoa may be found in the urine. They signify that the semen has been ejaculated, but in a wrong direction—toward the bladder.

SPERMATORRHŒA still remains to be discussed. If semen is voided in the course of a difficult movement of the bowels, spermatorrhœa should not be diagnosed. This is a very common, almost a physiological, condition. If a patient masturbates and then comes to you complaining of spermatorrhœa, do not call it by that name. The physician should not beat about the bush, but should plainly ask, Do you masturbate? Another patient, at the sight of an attractive woman or at the slightest sexual excitement, at once has an emission, or may have ejaculation without erection. This is the result of disturbances of innervation. Other forms of spermatorrhœa are rarely encountered. In most of these cases normal and regular sexual intercourse with a congenial woman is the best remedy, just as in psychical impotence the diplomacy of a clever woman usually brings about a quick and permanent cure.

The vulva has but few functional diseases, but its visible and tangible diseases are numerous. Herniæ deserve our first attention. An inguinal hernia which has descended into the labium majus is known as a *hernia labii majoris anterior.* A hernia which appears in the posterior part of the big labium after descending along the wall of the vagina in front of the broad ligament is designated as a *hernia labii majoris posterior.* If the hernia descends posteriorly to the broad ligament, it appears externally in the perineum, and is called a *perineal hernia.* If the contents of such a hernia is composed of small intestine, the diagnosis may readily be made by the tympany and reducibility. Even if the hernia is irreducible, the impulse on coughing is sufficient to draw a true conclusion. If the contents is formed by the bladder, so that no true hernial sac is present (by strict interpretation the condition is not a hernia, but rather a prolapse), the trouble is more important, as urinary symptoms arise. It must, therefore, not be omitted to empty the bladder by catheterization in order to avoid errors. In prolapse of the bladder the tumour will then diminish in size.

ELEPHANTIASIS of the labia is frequently not recognised by the inexperienced. If they see a large pedunculated mass, varying from the size of a fist to that of a head, hanging from the large labium, it is at once classed as a lipoma or fibroma. It is quite true that lipomata or fibromata of similar size and shape arise from the labia, but compared to elephantiasis they occur much more rarely. If the skin is intact, the structure of the tumour lobulated, and the consistency semi-elastic, the diagnosis of lipoma is justified. If the consistence is firm, the skin intact, and the mass is cystic in one or more spots, the diagnosis of fibroma may be made. If, however, the tumour is very large, the skin over either of the two neoplasms mentioned may ulcerate at the dependent portions of the mass, and the neighbourhood of the ulcerating area grow pigmented, fissured, thickened, and scaly. Over the central portions, however, the skin will remain normal, and both in lipomata and fibromata it will be freely movable over the tumour. In elephantiasis, the skin is thickened over the whole tumour, and at some spots, sometimes spots not especially exposed to irritation and friction, enormously thickened. The skin surface shows hypertrophied papillæ, and is warty, scaly, and covered by a cheesy secretion of strong odour. The tumour itself has a peculiar consistency. As the condition is due solely to a hypertrophy of the skin, and as the blood and lymphatic vessels take part in this hypertrophy equally with the connective tissue, the tumour in spots has firm strands running through it, while in other parts hard lumps may be felt. The whole mass may be somewhat diminished in size by squeezing the blood and lymph out of its vessels, thus, to a slight degree, simulating compressibility. The history should be of value. The swelling may have appeared after an attack of erysipelas, or erysipelas may develop frequently, and each attack be followed by an increase in size. All these tumours may readily be extirpated. If a tumour of larger size occupies the pubic region and is firmly adherent to the pelvic bones, it most probably is sarcomatous. If its consistency is firmer and the growth slower, it is most probably a chondroma. Neither are very uncommon.

Most important of the *cystic* tumours of the labia are retention-cysts of the glands of Bartholin or of the Bartholinian ducts. The strict circumscription, the fluctuation, absence of pain, inability to empty the swelling, all point to its cystic nature. If the swelling is elongated and spindle-shaped, the ducts have been occluded; if rounded or lobulated, the gland itself is the seat of the trouble.

Carcinoma of the vulva is uncommon. The diagnosis differs in no way from that of cancer in other parts of the body.

CHAPTER XXIX

DISEASES OF THE RECTUM

DISEASES of the rectum are numerous and important. At all ages and in both sexes we have frequent occasion to make rectal examinations, essential for the purpose of diagnosis.

The chief instrument used in surgical exploration of the rectum is the finger, for digital examination may clear up many doubts. A relaxed condition, strong contraction, or spasticity of the sphincter can be recognised. In prolapse, the mucous membrane feels unduly relaxed; in submucous cancer, it appears fixed. A carcinoma opposes resistance; moreover, its open, ulcerating spots, its extent and boundaries, may be palpated. The prostate, a portion of the urethra, the bladder, the seminal vesicles, are open to our touch. We can feel an extensive stricture, fistulous openings, polypi. In the virgin, we resort to rectal exploration in order to examine the uterus; likewise in a multipara suffering with cicatricial stenosis of the vagina. Periostitis of the sacrum may be directly palpated, pelvic abscesses are discovered, fractures of the pelvis or tumours of its walls may be felt, also tumours lying in the pelvis, etc. The finger discovers much more than the mirror.

Although I have repeatedly alluded to the method of Simon (insertion of the entire hand), a more detailed account of the method is required in this connection. Obstetricians have known for a long time that in deep narcosis the four fingers of one hand could readily be introduced into the rectum, in order to replace, let us say, a retroflexed pregnant uterus. Simon was the first to muster sufficient courage to introduce the entire hand.

After the sphincter has thoroughly relaxed under the influence of deep chloroform narcosis, first two and then four fingers of the hand are inserted through the anus. The fingers should first be thoroughly lubricated. The hand is then rotated from side to side in order to relax the sphincter still more, to permit the introduction of the thumb, thus admitting the entire hand. If the hand is now placed in the hollow of the sacrum, four fingers may be pushed upward into the narrower part of the rectum and, in some instances, may reach the beginning of the sigmoid flexure. This procedure has been practised in hundreds of cases. Its sole drawbacks are the incontinence which may persist on the following day and the slight fissures of the anus or sphincter which may result. The female pelvis is sufficiently wide to admit a hand of twenty-five centimetres circumference; the male pelvis may be unable to accommodate more than four fingers. The special advantages of this method of palpation need not be dilated upon. All the pelvic organs can be directly palpated. The operator takes advantage of this method when he introduces specula, which dilate the rectum and give him sufficient room to operate or to suture at his ease.

We are indebted to Simon for another diagnostic method. The large intestine may be explored with bougies as far as the descending colon if the curve of the sigmoid colon is not too great. It was formerly thought that the bougie could be pushed up into the transverse colon, because the tip of the instrument could be felt, through the abdominal wall, on the right side. As Simon has shown, this is not the true explanation; in such cases, the sigmoid is sharply curved, and the tip of the sound carries one limb of the loop to the right. It is important not only from a therapeutic standpoint, but also from the standpoint of diagnosis, to know that, according to Simon, the whole colon may be distended with water if the rectal tube is passed somewhat higher up into the bowel and water pumped in. Its diagnostic value at once grows clear if we think of its application in intestinal obstruction.

We must mention a method of examination recommended very warmly by Esmarch. The whole lumen of the rectum may be inspected if, with the patient in the knee-and-elbow position, the posterior rectal wall is drawn up toward the sacrum with a hollow, spade-like retractor.

Among the special diseases of the rectum, *congenital malformations* deserve the first mention.

The rectum may terminate blindly at its lower end, so that in the new-born the usual site of the anus is occupied by a small depression or fold. When the child cries or when the belly is compressed, the lower end of the gut, filled with meconium, bulges outward at this point. The condition is known as ATRESIA ANI. If the rectum terminates higher up, which may be recognised by the fact that no protrusion occurs during the examination, the resulting malformation is called

Atresia recti et ani. If the anus is developed, but leads into a short, blind sac, shut off above, the name of *Atresia recti* is applied. It should not be forgotten that the lower end of the intestinal canal may empty into the bladder or urethra, or, in the new-born of female sex, into the vagina. Consequently, atresia recti vesicalis, urethralis, or vaginalis may exist. In the last case, meconium flows out of the vagina; in the two preceding varieties, it is voided with the urine. In atresia vesicalis, the meconium is discharged only with the urine; in atresia urethralis, independent of micturition. The rectum may be shut off at the spot at which it normally ends, but empty, by means of a diverticulum, on the perineum, scrotum, or penis—in girls, on the vulva in front of the hymen. In all these cases in which the anus is imperforate, but in which the rectum opens at some abnormal site, the opening is known as an anus preternaturalis or fistula. As R. Frank has shown, the abnormal communications with the bladder, vagina, or urethra (internal anus preternaturalis, internal fistula) are due to the fact that the septum of Douglas has failed to unite with the perineal body. In other words, they are remains of the cloaca. The formation of external fistulæ (atresia perinealis, scrotalis, vulvaris) result from a persistence of the perineo-scrotal cleft. The atresia is to be regarded as a closure of the already developed rectum.

Although not found at birth, several diseases of the rectum occur in early infancy. In the first place, PROLAPSE of the rectum. We distinguish between prolapse of the mucous membrane (prolapsus mucosæ ani) and prolapse of the gut. In the latter, the whole thickness of the rectal wall takes part in the descent. If the anus forms the ring through which the prolapse descends, there is a certain difference between prolapse and intussusception.

Both in prolapse and in intussusception an inverted cone of the intestine descends out of the rectum. Both have a central opening. But in intussusception we can push a finger into the rectum along the entire circumference of the gut. In prolapse our finger is arrested at the anus, and we can see where the skin and the prolapsed mucous membrane are continuous. If the point at which the prolapse begins is higher up in the rectum, no such difference exists. The length of the intussusception is then the sole point of difference. If the rectum prolapses just above the sphincter, the condition really is one of invagination, but just to this the name of prolapse of the rectum commonly is applied.

A not uncommon condition is a POLYP of the rectum. This should be suspected if a child is known to suffer from frequent severe hemorrhages from the rectum. Digital exploration at once decides. If the pedicle has grown longer, the growth may project down to the anus and groove the fæces as they pass. If the pedicle is still longer, the polyp may protrude from the anus and be mistaken for a hemorrhoid. The presence of a pedicle is sufficient to establish a diagnosis. It is, of course, clear that polypi also occur in adults; their etiology, however, is obscure.

Demarquay observed the following case: In a woman of sixty-four years of age he saw a number of chestnut-sized bodies about the anus. In their midst a peculiar bright-red body, resembling the penis of a dog, projected outward. This was recognised as a fibrous polyp; and next to it were three smaller polyps. The patient confided to him that she had been forced, for several years, by her husband to submit to coitus per anum.

Prolapse of the mucous membrane alone, or of all the layers, are frequently called hemorrhoids. But the name HEMORRHOID should be confined to varices of the hemorrhoidal plexus—that is, to small nodules filled with blood, and in direct communication with the hemorrhoidal veins. Mere folds of mucous membrane are called by some clinicians *false* hemorrhoids.

True hemorrhoids are circumscribed, bluish lumps, which bleed readily and empty on pressure. According to their point of attachment, they are classified as external and internal, the point of division being formed by the sphincter ani. The symptoms which accompany the trouble—discharge of mucus, catarrhal proctitis, pain, frequently profuse hemorrhage, itching about the anus—can be readily understood. The condition grows serious when a hemorrhoid becomes inflamed and strangulated. Fever, hiccough, and vomiting may result, so that the whole picture resembles that seen in strangulated hernia. When seeking for the *cause* of hemorrhoids in a given case, inquire whether the patient is constipated, and has been in the habit of using strong purgatives. The pelvis must be examined for tumours or for enlargement of some of its organs, among which we include pregnancy. Finally, stricture of the urethra or vesical calculus must not be forgotten, for the effect of abdominal pressure exerted during the severe straining in obstructed micturition may cause stasis in the hemorrhoidal veins. In many cases careful examination will fail to detect any cause.

CARCINOMA of the rectum also presents itself to us under the name of hemorrhoids, either because the patient believes this to be the cause of his trouble or because another physician has consoled him by calling the disease by that name. The symptoms and the difficulty experienced in making the diagnosis depend upon the site of the neoplasm. Epithelioma of the anus, recognised by its hardness and ulcerating surface, can be seen externally. If the carcinoma develops in the lumen of the rectum, it may be thoroughly explored by the examining finger. The findings are a sharply circumscribed tumour, either in the shape of a flat surface, of a semicircular, or complete ring of growth embedded in the wall of the gut. Its free surface is ulcerating; the whole picture is unmistakably that of an epithelioma. Scirrhous cancer also occurs; it is submucous. If the mucous membrane is still smooth, normal to the touch, but adherent at one spot to the hard submucous growth, the suspicion of cancer should be very strong, and the diagnosis can be made by excluding other similar processes. In many cases, if the cancer is situated higher up, the growth does not give the impression of a platelike surface, but rather of a mass the size of a child's fist, projecting into the lumen of the rectum. This mass is characterized by its hard, indurated base, and its deep fissures and ulcerating surface. Finally, advanced cases are encountered in which the whole rectum has been changed into a sloughing, cancerous mass, and into which the finger can scarcely be pushed through the narrowed and distorted lumen. In most cases a single rectal examination will suffice to make the diagnosis of cancer. But, as cancer of the rectum now is capable of the most radical surgical interference, the examination should also allow us to judge whether local extirpation is feasible or not. It is important to determine whether the upper margin of the growth can be reached, and whether the tumour can be pulled downward. If the disease has existed for a long time, and has extensively spread, other symptoms are found in rectal cancer. Large, infiltrated retroperitoneal glands can be palpated; the heterologous character of the growth is established by its extension to neighbouring tissues. Involvement of the sacrum fixes the growth to this bone. If the cancer spreads to the bladder, strangury, cystitis, perforation with gangrenous inflammation of the bladder, result. Neuralgic pains in the lower limb point to an involvement of the nerve-trunks. All grades of stricture naturally complicate both operable and inoperable cases.

If the cancer is situated high up in the rectum, the rectum must be exposed to the eye by means of a speculum, or palpated, in narcosis, by Simon's method. The symptoms are quite characteristic. Chronic intestinal stenosis—which may be recognised by "lead-pencil" stools and habitual constipation—is the first symptom. After this has existed for some time, a marked increase of the stenosis is noticed; blood is passed in the stools and necrotic masses of tissue come away with the fæces. After large masses of the growth have been passed, the stenotic symptoms show a slight remission, but they again gradually increase. Defecation is frequent; the stools are thin, contain blood, and are reduced in amount.

Other conditions may cause symptoms of a somewhat similar character. These include all varieties of STRICTURES OF THE RECTUM. They may be due to internal ulcers and inflammatory processes, which are characterized by the formation of cicatrices (absent in cancer). Symptoms in other parts of the body—for instance, signs of syphilis elsewhere—are of corroborative value. The course also differs from that of cancer. In benign stricture, the symptoms appear slowly during or after an attack of ulcerative nature; in cancer, the symptoms of slight stenosis alone are found, while the growth remains confined to the submucosa; after it breaks through, ulcerative symptoms appear —hemorrhage and sloughing.

The more doubtful cases are those in which the symptoms of stenosis and ulceration are observed at one and the same time, found both in ulcerating cancer or in inflammatory stricture, still in the stage of active ulceration. The latter variety are found in follicular ulceration of the rectum, in dysentery, tuberculosis, syphilis, and in gonorrhœal proctitis by direct infection. Especially in syphilis and in dysentery, more rarely in tubercular ulceration, superficial erosions go hand in hand with a firm infiltration of the deeper layers and consequent stricture. The discharge of blood and *pus* points to ulceration. Direct inspection of the mucous membrane is then resorted to. Multiple ulcers or erosions are seen; their nature must be determined from their appearance, course, etiology, and the general make-up of the patient.

Dysenteric ulcers are usually recognised by the fact that the patient is suffering with dysentery. Their site is the upper portion of the rectum. *Tubercular ulcers* exist in phthisical patients. The ulcers are characterized by small, gray or yellowish breaking-down nodules, which are situated upon the irregular undermined edges. At some spots there are small, gray or yellowish nodules, and at others small, sharply circumscribed ulcers, due to softening of these nodes. *Syphilitic ulcers*, which cause the most extensive destruction, are accompanied by symptoms of syphilis in other parts of the body. Locally, gummata can be demonstrated. They are most apparent at the margins of the process, therefore, especially at its upper limit, as the infection extends upward. On other peripheral spots, however, these small nodes, which reach the size of a pea and are round and dark red, may be found. While nodules are breaking down and discharging their brown-red contents, others have already formed sharply circumscribed ulcers. If the involvement is extensive, the landmarks disappear, and healthy spots of mucous membrane can no longer be seen between the numerous ulcers.

Polyposis recti is not uncommon. The wall of the rectum is found covered over a large extent by discrete pedunculated growths, which are soft, and show no tendency to ulcerate. The mucous membrane is otherwise normal.

FISTULÆ IN ANO are next in order. To determine whether a fistula, through which neither gas nor fæces are passed, is complete or incomplete requires the use of a probe. This really covers all the essentials in the diagnosis of this condition. Each case demands careful examination. One point deserves mention: true fistulæ have their internal opening immediately above the sphincter. If the finger is passed into the rectum and it is found that the probe can pass higher up along the rectum, or that two or more fistulous tracts, parallel to each other, run farther up, a more deep-seated cause should be looked for. As a rule, it will be found to be caries of the inner surface of the sacrum, which can be recognised either by an elastic swelling behind the rectum or by local tenderness. In women, parametric abscesses must be taken into account.

Spasm of the sphincter and *fissura ani* remain to be dealt with. If the patient complains of excruciating pain, which persists for hours after each movement of the bowels, the disease is sufficiently characterized. Local examination may show a small tear not broader than a hair in the folds of the anus, or a small ulceration, or nothing but a severe spasm of the sphincter. Great pain is caused if we attempt to introduce the finger into the rectum.

A woman who suffered great pain on defecation travelled all over Europe to visit the various gynecologists. The case was always diagnosed as retroflexion of the uterus. When she visited Linhart, he recognised the trouble as one of fissure ani (without examination) by the patient's account of severe pain after each stool. He cured her! This story is worth remembering.

As a rule, diseases of the rectum cause great suffering. An old philologist, out of whose rectum I ladled huge masses of fæces, and thus almost renewed his life, sighed thankfully, "How right was the physician in Plato's Symposium, who defined the healing art as the science which deals with the proper filling of the organs!" Therefore, examine carefully.

CHAPTER XXX

INJURIES OF THE HIP-JOINT

THERE are cases in which the diagnosis between fracture of the neck of the femur, contusion of the hip-joint, and dislocation of the hip is difficult to make. I myself have had to deal with more than one such instance. The most careful and detailed examination, combined with thorough weighing of all the evidence obtained, was necessary. But, on the other hand, I am loath to say that in the clearest cases physicians still make frequent errors, which result in great damage to the patients—usually poor people. They are guilty of an error in logic, against which Pitha warns them in the following words: "Do not rest satisfied with the vague idea of *a* luxation; put the precise question to yourself, and find a *definite* variety of dislocation before you rest content." Pitha grasped at the very root of the evil. In practice, we always find that the physician in question was satisfied with the diagnosis of *a* dislocation. This clearly is modesty carried to the extreme. As much of the anatomical changes as are required to aid in the diagnosis are readily remembered by all. If the picture which we demonstrate on the skeleton is forgotten in practice, it may be recalled in the following way: Take a pencil and let it represent the leg in the vertical position. Stick a needle into the lower end in an anteroposterior direction; it is to represent the axis of the foot. Into the upper end stick a needle directed from right to left, with its free end slightly raised, to represent the neck of the femur, and, if you care to, you may add a ball of wax, to indicate the head. Then place

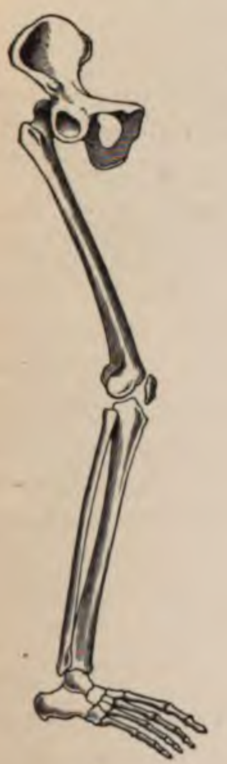

FIG. 42.
Luxatio ischiadica.

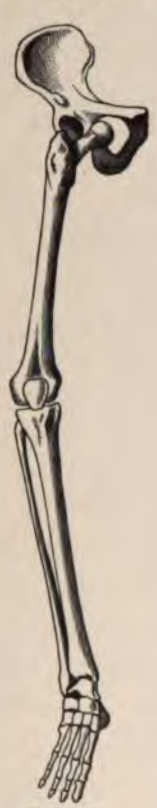

FIG. 43.
Luxatio obturatoria.

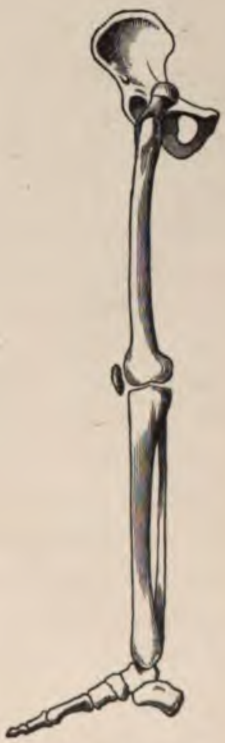

FIG. 44.
Luxatio pubica.

the figure so that it corresponds exactly to the right or left leg. If the pencil is now rolled between two fingers, rotating about its long diameter, we see the following: As the head of the femur moves backward the anterior end of the foot's axis rotates inward; the extremity is rotated inward. If the head is directed forward, the foot turns out; the extremity is rotated outward. In other words, *in posterior dislocations the leg rotates inward; in anterior dislocations, outward.* If this seems too pedantic, bear in mind that you may find yourself in the unpleasant position of explaining this fact to an ignorant and refractory colleague.

Remember, also, that in dislocations in which the head lies above the acetabulum, the extremity is shortened; in those in which the head comes to rest below the joint cavity, the leg is lengthened. Finally, do not forget that the trochanter points to the situation of the head of the bone. If the trochanter is properly grasped, the direction of the head can be determined by projection.

Henry Bigelow, whose well-illustrated and useful book on Dislocations of the Hip-joint has been translated into German and is widely read, divides dislocations into regular and irregular forms. Regular luxations include those in which the ligament of Bigelow is intact, or only one of its bands has been ruptured, so that the position of the extremity is still influenced by the tension that this structure exerts. Irregular luxations are those in which the entire ligament is torn away, so that the dislocated head blindly follows the force exerted, without modifying influences. No matter if the expression "regular" and "irregular" is a happy one or not, it at least corresponds to the salient features of the dislocation. Irregular luxations presuppose the exertion of enormous forces, consequently they are uncommon. Typical dislocations are frequent. They require a definite, rational method of reduction. These accidents occur chiefly in the country and among the working classes. I accept the classification of my teacher (v. Dumreicher), who was the first, in Germany, to recognise the methods of reduction. This classification divides dislocations at the hip into only three varieties: one posterior and two anterior. *In posterior dislocation*, the head of the femur rests upon the body of the ischium—therefore known as luxatio ischiadica—the limb is rotated inward, adducted, flexed, and shortened, so that the foot of the affected side comes to lie above the healthy one, and also crosses it. Practically it is unimportant whether the head assumes a higher or a lower position; it merely presupposes a greater or lesser degree of displacement. The thigh is flexed,

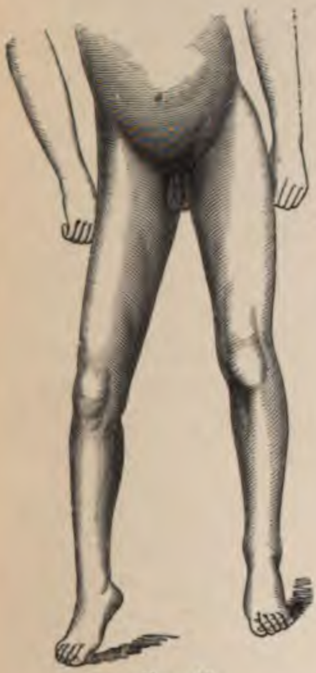

FIG. 45.
Luxatio obturatoria.

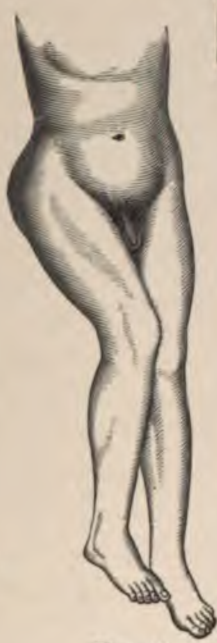

FIG. 46.
Luxatio ischiadica.

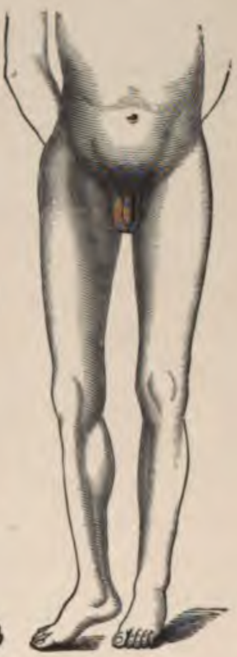

FIG. 47.
Luxatio pubica.

adducted, and rotated inward in all posterior dislocations. *In anterior dislocation*, the head rests either upon the horizontal ramus of the pubic bone—luxatio pubica—or upon the thyroid foramen—luxatio obturatoria. In both, the lower extremity is rotated outward, but other differences are great. In the pubic dislocation the limb is materially shortened, extended at the hip, and the head of the femur is either *seen* or *felt* upon the horizontal ramus. In obturator dislocation, the extremity is not visibly shortened, the hip-joint is slightly flexed, and the head is *not palpable*. It lies deeply in the mass of muscles, and even the *trochanter is no longer visible*, as it now rests in the acetabular cavity. At the site at which the trochanter usually forms a prominence there is a hollow, and the mere sight of this depression, combined with the above-mentioned pathognomonic position, suffices to make the diagnosis.

We have discussed dislocations before other injuries, because they furnish the most definite symptoms; they are the completed pictures with which the other injuries are to be contrasted and compared. As the position of the limb is the most striking symptom, we will take it as our guide.

Let us assume that the injured person lies before us with the extremity flexed, adducted, and rotated in; these are the symptoms of a dislocation onto the ischium. In fractures of the neck of the femur the thigh may, but very rarely does, assume this position. If I had not seen it with my own eyes I would scarce have believed that the resemblance could be so close. The following signs will be found of value, in the differential diagnosis, on those occasions on which it may prove difficult:

1. In subjects of advanced age, a fracture of the neck is very probable, a dislocation unlikely.

2. The probability in favour of fracture is increased if ecchymoses occur over the trochanter, for these are most often due to a fall on the trochanter—which is a common cause of fracture of the neck.

3. The chances are still more in favour of fracture if the trochanter seems broadened. It would be a sure sign were it not for the difficulty in judging such an increase by palpation. Therefore, caution demands that this symptom should not be regarded as pathognomonic. The same remark applies to crepitus.

4. In fracture of the neck the gluteal region remains unaltered; in ischiadic luxation its outline is changed. The upper portion of this region is more flabby and relaxed, the lower portion bulges outward. (In cases of long standing the findings may be reversed, as the head secondarily assumes a higher position.)

5. In thin subjects, the region of the acetabulum feels *empty* in dislocation. If the resistance of the tissues immediately in front of the horizontal pubic ramus is tested by pressing downward with the fingers, the bony resistance normally offered by the head is found wanting.

6. When the head is felt, on rotation, rolling, deeply among the muscles of the gluteal region, it is *decisive.* If the patient is thin, it is impossible to mistake or overlook the head of the femur; if the patient is fat, it is more difficult to feel the head, but we must persevere until a positive conclusion has been reached.

If any doubt remains, examination under anæsthesia must be employed. The spastic resistance which persists in dislocation disappears to a great extent in fracture, so that the limb can be straightened without exertion and without producing a snapping sensation.

Let us assume another case: The patient lies helpless with his limb everted, the knee- and hip-joints extended, with active motion impossible. This position, if shortening were also present, corresponds to a pudic dislocation. This variety may, however, at once be recognised or excluded. Merely palpate the body of the pubis and determine whether the head of the bone is there or not. Unless the swelling hides it from our sight, inspection alone will serve to recognise it.

The head is not found there, but lies immediately beneath the anterior superior spine of the ilium. The trochanter, as a result of the outward rotation, is somewhat posterior. This corresponds to an irregular dislocation—the *supracotyloid* variety, of which a number of cases are on record. If we assume that the head can not be felt, we are confronted by a fracture, by a contusion in which the eversion is accidental, or by a special variety of fracture of the pelvis.

This form is still known by the name it received from Malgaigne—double vertical fracture. A narrow but long piece of bone is broken out of the pelvis; it includes that part of the bone on which the acetabulum is located. The fragment rotates, to some extent, upon its long diameter, so that the limb is everted. If we follow along the iliac crest we come to a break in continuity, and the narrow middle fragment is found movable.

The possibility of fracture of the neck of the femur and of contusion have not yet been excluded. Positive knowledge that the patient's legs were of equal length before the injury was received, would at once exclude contusion if shortening could be demonstrated. But as this is not always to be obtained, measurement of the length of the limb is not sufficient. Determine by measurement whether the trochanter is higher up or not. If the region is much swollen, and shortening inconsiderable, this symptom is inconclusive, and further signs of fracture must be looked for. In addition to eversion, pain, and loss of active motion, we find: 1. Crepitation, which, if well marked, is conclusive evidence in favour of fracture. 2. A peculiar phenomenon on rotating the thigh. If the neck of the femur is intact, each portion of the trochanter describes an arc whose radius is equal to the distance between a given point and the centre of the head of the bone. If the neck is fractured, the trochanter rotates about the long axis of the thigh, and the points describe smaller arcs. I may add, however, that it is not easy to elicit this sign. 3. The proof is more readily obtained if, after the pelvis is properly fixed, the relative position of the trochanter can be changed by alternately pushing the thigh upward and pulling it downward.

If the fracture is impacted, many of these points of differentiation no longer hold good. Experiments performed by Heppner and others have shown that impaction of the fractured neck into the trochanter results when the fracture is caused by a fall upon the trochanter. If the force acts in the direction of the long axis of the thigh, the neck is fractured, but without impaction. If in doubt whether the case is one of fracture or contusion, bruises in the trochanteric region and broadening of the trochanter would favour the diagnosis of fracture, and the case should be treated as such. In the course of a few days the impaction yields and the diagnosis is then more surely arrived at. From a medico-legal standpoint this is important.

In the days when crinolines were worn, a well-known member of the demi-monde knocked down an old beggar with her crinoline. A fracture of the neck of the femur resulted. The case did not come before the courts; but, nevertheless, the question arose whether the beggar had sustained his injury by being thrown over, or whether the fracture had been caused by a misstep resulting from his attempt to get out of the way—i. e., through the pressure of the body weight suddenly brought to bear on the weakened neck of the bone, so that the fall was due to, and preceded by, the fracture. In similar cases, experience at the bedside and experiments would justify us to decide that the result was due to a fall, if bruising of the trochanteric region and impaction were subsequently found.

Obturator dislocation is characterized by immobility in the flexed position, with adduction, eversion, and a deep depression in the region of the trochanter. These signs are distinctive of this injury.

Dislocation of the head vertically downward upon the tuberosity of the ischium produces symptoms which also are distinctive. The most striking symptom is the *increase in length* of the limb, which is stiffly held in a median position. The head is not palpable, and the resistance of the neighbouring parts does not permit us to determine whether the acetabulum is empty or not. But the low position of the trochanter, combined with the rigid position of the limb, can be accounted for only by this variety of dislocation.

The anatomical reasoning which has been applied to traumatic hip-joint dislocations can not be applied to CONGENITAL DISLOCATIONS of the hip. This condition is found more frequently in the female sex. It may be unilateral or bilateral, and is first recognised when the child begins to walk. The head is outside the acetabular cavity, and is located upon the dorsum iliæ, where it may plainly be felt. *In spite of this the position of the limb is not pathognomonic.* The gait, however, is characteristic, and the phrase "duck's waddle" (Entengang) defines it, if the condition is bilateral. Marked lordosis, and, as a result of this, strong tilting of the pelvis and broadening of the upper portion of the gluteal region, are the most striking symptoms when the child is standing. When the child takes a step forward, the trunk sinks and inclines toward that side on which the leg is being planted. If the child is laid down, the thigh, especially if flexed and adducted, may be moved up and down along the pelvis. These are the principal symptoms of this common deformity.

CHAPTER XXXI

INFLAMMATORY PROCESSES OF THE HIP-JOINT

THE existence of a coxitis is frequently not recognised by physicians. It is true that the symptomatology of the disease is varied, and that the pathognomonic position of the limb may also vary; but, for that very reason, the essential diagnostic sign should not be forgotten. Therefore I will state at the outset, that this sign is *fixation of the joint by muscular action in the pathognomonic position.* These words should be learned by heart, for they include the whole lesson in a nutshell. Every experienced practitioner makes the diagnosis of coxitis as soon as he sees the position of the extremity, after he has determined that the pelvis *accompanies* passive movements of the thigh. To do this, place one hand upon the pelvis and with the other extend the thigh.

To prove that the fixation is due to muscular action is best accomplished by making small and cautious efforts at motion. These, as a rule, can be freely made unless the sensitiveness is extreme. The signs of rigidity are accompanied by the usual symptoms of inflammation. These are, swelling about the joint, tenderness, especially tenderness in the joint when the thigh is pressed upward against the acetabulum, etc. Of special importance is the mysterious referred pain—

i. e., pain felt in the knee, and sometimes at the ankle. It is readily understood that in a disease whose course extends over many years, various complications should arise, but the simple rule, above expressed, nevertheless holds good.

The pathognomonic position of the joint corresponds in most cases to the *position of rest*, and consists of moderate flexion, abduction, and rotation outward. This position is only evident when the pelvis is normally placed. By flexing, tilting, and rotating the pelvis, the patient may mask the position so that the two limbs lie parallel upon the bed. The compensatory movements take place in the healthy hip-joint.

By flexion is understood a forward movement of the pelvis about its transverse axis; by tilting, a downward movement about its antero-posterior axis; by rotation, a twisting about its vertical axis.

How is the pathognomonic position masked? As above, we take for granted that the thigh and pelvis form, so to speak, a jointless unit. In order to overcome the flexion, the patient must *flex* his pelvis till the limb rests upon the support. This movement betrays itself at once by the compensatory lordosis of the lumbar spine. To counteract the abduction the patient must *tilt* the pelvis downward upon the diseased side; this is at once recognised by the fact that the limb seems longer. Formerly, before Bonnet's time, it was thought to be actually lengthened, but by comparing the spines of the ilia on both sides, it is seen that the spine on the diseased side has descended a distance equal to the apparent lengthening of the affected limb. By comparing the measurements of the two sides from the iliac spines downward, it is noticed that there is no lengthening, but in the advanced stages an actual shortening of the diseased extremity. Finally, the patient can conceal the eversion by *twisting* the pelvis—i. e., by a movement in the healthy joint about an axis parallel to the long axis of the body, just as the patient turns when changing from a dorsal posture to a position on his side. This movement is betrayed by the hollow found beneath the pelvis on the diseased side. As a rule, the patient omits this third corrective change and contents himself with flexion and tilting of the pelvis. In order to bring the pathognomonic posture into evidence, the following manœuvre must be employed: Grasp the diseased limb and gently increase the flexion until the normal angle between pelvis and spinal column has been restored. This is attained when the back rests upon the support and the lumbar lordosis has been effaced. The thigh is then abducted until the spines of the ilium are at the same level, and finally, if the patient has masked the rotation, evert the extremity until both sides of the pelvis rest equally upon the examining table.

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In Fig. 48, the patient lies with a lordosis of the spine, and with strongly tilted pelvis, in order to place the thigh flat upon the table. Fig. 49 shows the trick unmasked, and also gives a view of the pathognomonic position in coxitis.

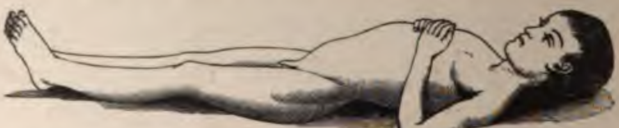

FIG. 48.—Lordosis and tilting of the pelvis in coxitis.

Fig. 50 represents a patient with the left half of the pelvis markedly tilted downward in order to bring the two legs parallel. In Fig. 51, after the position of the pelvis has been corrected, the characteristic abduction at once becomes apparent.

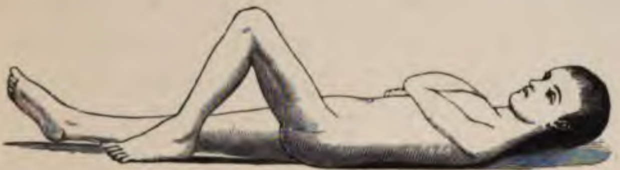

FIG. 49.—Flexion and abduction in coxitis.

Rotation of the pelvis can be demonstrated in a more precise fashion. In the normal position a frontal plane passes through the superior iliac spines of both sides. When the pelvis is rotated, a plane passing through these points no longer is frontal in its direction.

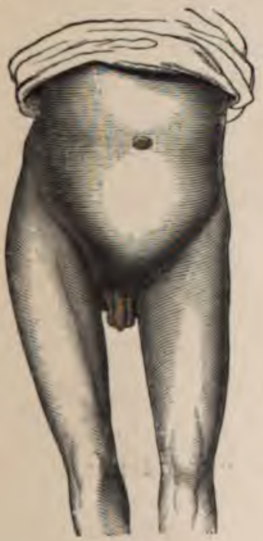

FIG. 50.
Tilting of the pelvis in order to bring the limbs parallel.

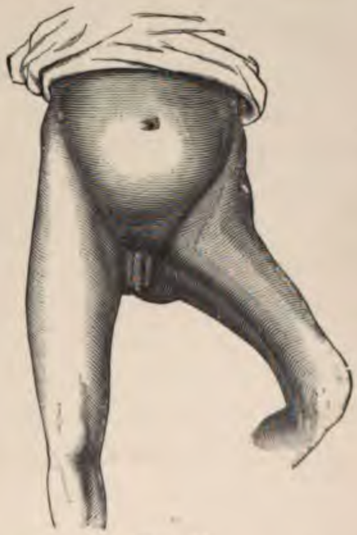

FIG. 51.
Correction of the compensatory tilting, showing the resulting abduction.

It has been mentioned above that the increased flexion of the pelvis is recognised by a lordosis in the lumbar spine. This means that the position of lordosis attracts our attention to the increased angle of flexion. The increased flexion can be *directly* recognised by the fact that the sacrum no longer rests horizontally upon the table, but forms an angle, of varying degree, with this support.

Examine Fig. 49 and ask yourself: If the left leg were flexed to a right angle, what movements would the pelvis make upon abduction and adduction? Evidently, a movement of rotation. If Fig. 48 is kept in view, the question reads: If the left leg is extended, what movements would the pelvis undergo on abduction and adduction? The answer naturally is, Tilting upward and downward. In other words, the movement of the pelvis depends upon the position of the limb. If I abduct and adduct the thigh, which is flexed at an angle of ninety degrees, points along the thigh and also points taken on the pelvis (which moves with the thigh) move in a plane which is vertical to the axis of the body—i. e., referred to the pelvis, rotation. If I abducted and adducted the extended thigh, points taken along the thigh, and consequently points on the pelvis, move in a frontal plane—i. e., referred to the pelvis, an upward or downward tilting.

What movements must I make, with the thigh flexed at a right angle, in order to tilt the pelvis up and down? Whoever does not find the solution to this question at once does not understand the condition.

In another set of cases a different pathognomonic position is encountered—flexion, adduction, and inversion. The sick child draws up the diseased limb upon the healthy one, using the latter as a support, exactly like a splint.

Here we must determine whether this position was primarily present or only appeared later in the course of the disease. There are cases of coxitis in which this position is assumed from the outset; in others, the first-mentioned position of abduction and eversion later is replaced by adduction and inversion. A rule that the so-called stage of apparent lengthening in the first position is followed by a stage of shortening in the second position, has been formulated. For many cases this holds good, but not for all.

In addition to the two typical positions of the limb, other postures are assumed in the course of some cases of coxitis. For instance, the flexion may increase to a right angle, or even to an acute angle. The abduction may grow so excessive that the thigh stands out at right angles from the trunk, and in other cases, although the flexion is scarcely noticeable, the eversion is strongly marked. In all cases, however, the muscular spasm, the fixation by the muscles, remains the most important symptom.

Next in importance to the above-mentioned symptoms is the proof that *actual shortening* has taken place. In the typical coxitis of childhood the shortening occurs early in the disease. It may be due to the following causes:

1. The trochanter may move upward if the neck of the femur assumes a more horizontal position.

The muscles which run from the pelvis to the femur work in two ways: they pull the femur upward, and also press it inward toward the acetabulum. As the head and neck of the bone are softened by the inflammatory process, and the muscles about the joint continue to press the head against the acetabular cavity (while they at the same time fix the joint), the neck gradually diminishes and grows shorter. The trochanter meanwhile is pulled up by the vertical component, so that the neck lies horizontal or even at an acute angle to the shaft, and is at a higher level than the head of the bone. This mechanism of shortening is uncommon. The same result is more commonly due to a destruction of the head and neck by the disease.

2. The shortening may result from the so-called "wandering acetabulum." This is nothing more than an extension of the acetabulum in an upward and backward direction, as a result of the pressure exerted by the head of the femur. The shape of the cotyloid cavity changes from circular to elliptical, with its long axis directed from below and in front, upward and backward. The head, and with it the trochanter, move upward.

3. Secondary dislocation, either upward or backward, may have occurred. Gross changes may have taken place at the upper extremity of the femur: distortion of the head, shortening of the neck, or complete erosion of both, so that the trochanter now forms the upper extremity of the bone.

It is safe, as a rule, to assume a secondary dislocation, if the shortening is considerable; the case is positive, if the dislocated head can be felt outside of the joint. The secondary dislocations resulting from coxitis differ greatly from dislocations produced by trauma. The cause of this difference is recognised if the records made at the autopsy table are perused. One account reads, that the head and neck have entirely disappeared, that the trochanter has been dislocated upon the ilium. The limb is rotated inward. In other cases, in which the destruction has been almost as extensive and the trochanter moved upward, the thigh was found everted. In contrast to these cases, others are seen in which the head is almost entirely preserved, the neck somewhat shortened, but both plainly felt in the gluteal region.

Measurement of the limb is of the utmost importance in hip disease. It is the scale by which we judge the progression of the changes which take place in the bony constituents of the joint. It is both the proof of the presence and of the amount of destruction already done by the tubercular process, and is of decisive diagnostic importance in differentiating hip disease from other conditions. *Hysterical* women may imitate the picture of coxitis in all its details. The pathognomonic position, the muscular spasm, the tenderness, are all there. The condition may persist for months, but shortening does not occur. Only by continually taking measurements are we able to satisfy ourselves that no destructive process is going on in the joint. The condition is simply a neurosis. A deep *abscess*, which bathes the joint capsule, may produce complete rigidity of the joint. Even if the pathognomonic position is not quite characteristic, we suspect an atypical case of coxitis. It is only by proving that no shortening takes place that an important point in establishing the differential diagnosis is obtained.

Fixation of the joint is not peculiar to hip disease alone. As has just been stated, it may be due to an abscess in the vicinity of the joint. In other conditions it is also found, although in a modified form. In *psoitis* the joint is flexed, and there is also a trace of external rotation. If the pelvis is grasped and the leg extended, the pelvis follows this motion; if attempts at inward rotation are made, the same holds good, but in all other directions movement is unimpaired. In *inflammation of the inguinal glands*, it occasionally happens that motion is impaired in all directions which increase the tension of the inflamed parts; but all other movements are free. In contrast to the above, all observers agree that in inflammation of the bursa beneath the psoas—*bursitis iliaca*—extension of the hip is not impaired, although the bursa must surely be compressed as a result of this motion.

This condition may be diagnosed if a square, sharply circumscribed and fluctuating swelling is found in the inguinal region. The swelling is cut in two by the ilio-psoas, but communicates beneath the muscle. The swelling may grow so large that it extends to the spine of the ilium.

If the diagnosis of hip disease has been made, the next question to decide is, where the primary focus was located. The starting-point may have been in the acetabulum, in the capsule, or in the femur. We are, however, rarely able to determine this point. Only in those cases in which a severe exacerbation is followed by a spontaneous dislocation, which is very readily reduced, but recurs upon the slightest exertion of force, or even as the result of muscular action, are we justified in suspecting a distention of the capsule with effusion. This would point to involvement of the capsule, but does not exclude a focus in the bone.

Coxitis rarely occurs in adults. The symptoms are the same as in children. The increasing destruction, especially the abscess formation, points to the tuberculous nature of the trouble. Severe pain in the early stage confirms it. Less severe grades of destruction (shortening of the limb) also occur in *coxitis deformans*, but the fixation and the pain are less pronounced than in the tubercular form.

In the last few years a condition which was formerly considered a *healed* coxitis has been noticed. This condition has nothing in common with tubercular hip disease. The leg is shortened, walking is painful, the pelvis moves with the leg in abduction; the onset is gradual. If the leg is everted, it is still easier to confuse the condition with a mild, healing coxitis with atypical position of the leg. We now know that these cases are the result of purely mechanical changes. The chief character of the change is a more horizontal position assumed by the neck of the femur (consequently true shortening). The arc of abduction is decreased. The condition is analogous to the static deformities (genu valgum and varum, etc.), and is known as COXA VARA.

CHAPTER XXXII

INJURIES OF THE KNEE-JOINT

THE knee-joint is very superficially situated. This, combined with its massive structure and the characteristic shape of the bones which enter into the formation of the joint, make it more accessible to palpation than any other joint in the body. The physiological movements which here take place are so complicated and yet follow well-understood laws so closely, that disturbances of motion can be interpreted without difficulty. All these points taken together make the diagnosis of diseases of the knee-joint uncommonly easy, and therefore favourable to the beginner, because he can readily acquire insight into the method of diagnosis and gain confidence in his own efforts. Open injuries demand examination by means of direct inspection and palpation; they are therefore only of casuistic interest. Very little can be said of them in general. Subcutaneous injuries, dislocations, and fractures are of interest because they follow fixed rules. The diagnosis is never difficult, even if considerable swelling obscures the whole region. Palpate thoroughly, for only in exceptional cases with excessive swelling can any difficulty arise. If the injury is recent, and no swelling has formed, the changes are unmistakable. We are able to see and feel them, and the picture allows of but one interpretation.

FRACTURES OF THE PATELLA can not be overlooked, for the line of fracture can be both seen and felt, no matter whether it is transverse or longitudinal, whether broad or narrow. Dislocations of the patella can not fail to attract attention. In the lateral variety the intercondyloid fossa (the trochlea) is found empty; the patella lies upon the external condyle of the femur; the leg is flexed and rotated outward. In vertical dislocation of the patella, the edge of the bone stands out like a ridge on the anterior surface of the joint. The patella has made a twist of ninety degrees, and no longer lies with its surface, but only with its edge, applied to the joint surface of the femur.

DISLOCATIONS OF THE TIBIA in the recent state present a striking picture. It is, as a rule, the femur and not the tibia which passes through the capsular rent, and is displaced; but, as the tibia is the peripheral bone, it is said to be dislocated. We rarely see such a dislocation. I myself have seen but few; but it is easy to imagine the condition or to produce it on the cadaver. Lateral dislocations are described with much enthusiasm by observers fortunate enough to see them. The axis of the leg is parallel to the axis of the thigh, but at the knee a broken line ╵─╷ is produced. The joint surface of the tibia is recognised through the tense and thinned-out skin which at each moment threatens to give way. The surface is exposed so that the finger may be placed upon it, or, as Pitha used to say, so that a glass of water could safely stand upon it. On the opposite side the end of the femur is readily palpated. The patella lies transversely. Who can mistake this picture?

Anterior dislocations, by far the most frequent, are not as striking, but still sufficiently so to be always readily recognised. The anterior surface of the joint is altered by the position assumed by the patella. This bone lies almost upon the upper surface of the tibia, while above the patella is found a deep semilunar fold of skin with its convexity directed upward. Behind, the posterior surfaces of the condyles of the femur are prominent, and can be readily grasped.

The rare posterior dislocation causes the posterior edge of the tibia, and also the posterior portion of its joint surface, to become prominent. The patella is firmly pressed against the femur, and the long axis of the leg is behind the long axis of the thigh.

In order to make the deviation in the longitudinal axes more prominent, it is evident that in lateral dislocations the limb must be looked at from in front, in anterior or posterior luxations from the side. The extremely infrequent rotatory dislocations of the tibia are recognised by the rotation of the foot. The limb is not broken, yet the leg is twisted on its axis, so that the fibula lies posteriorly, the inner edge of the tibia anteriorly. The patella is vertically rotated, its tendon is tightly stretched, the joint fixed.

The infrequent *dislocation of the fibula* at the upper tibio-fibular joint is readily recognised. If the dislocation is in an anterior direction, the head of the bone lies upon the ligament of the patella. It is seen to be continuous with the shaft of the fibula, and to have the biceps tendon, which now runs in an arched direction, inserted into it. If a posterior dislocation occurs, the deformity is no less typical. A considerable bony projection is felt posteriorly, which is continuous with the fibula. The biceps tendon can be traced to this projection. In both varieties a deep hollow is found at the normal site of the tibio-fibular joint.

Rupture of the quadriceps tendon also gives a clear picture. If the tendon is torn above the patella, a furrow can be felt between the two ends. If the patellar ligament is ruptured, a furrow is found below the patella, and that bone is drawn upward.

FRACTURE OF THE LOWER END OF THE FEMUR causes even more marked displacement and greater shortening than that due to dislocation. The serrated edge of the upper fragment can be felt above the patella, and may perforate the quadriceps tendon or the skin. The uneven surface of the lower fragment may be palpated in the popliteal space. Fracture of one condyle of the femur can not be recognised by inspection, but by palpation, and by noticing the abnormal mobility, the diagnosis can be readily made. In the so-called T-shaped fractures of the lower end of the femur, in which the line of fracture runs transversely across the long diameter, and in which the lower fragment is divided by a vertical fracture separating the external and internal condyles, the separation from the rest of the bone can not be overlooked. The vertical fracture may more easily escape notice. Never forget to test the mobility of one condyle upon the other.

Only one variety of subcutaneous injuries of the knee-joint can not be recognised by direct examination, and must be diagnosed by the combination of symptoms. This is RUPTURE OF THE LIGAMENTS OF THE JOINT.

I observed the following case at v. Dumreicher's clinic: A man, of about thirty years of age, strong, and tall of stature, was engaged in pulling a hand-cart. He was caught between two street-cars and struck by one of them. His leg was firmly fixed, the shock bending his body in a forward and outward direction with such force that he fell down. He was at once brought to the clinic. The left leg was somewhat rotated inward, and also slightly dislocated backward. This displacement could be reduced without exerting any force. The joint could be overextended, therefore the crucial ligaments were torn; strong abduction of the leg was possible, therefore the internal lateral ligament was no longer intact. The leg could be rotated about its own axis, the external condyle acting as the fixed point, and as adduction was resisted, the external lateral ligament evidently was not torn through. Later, I saw a similar case in private practice. A twenty-year-old girl, carrying a burden on her shoulders, sat down on a mound by the roadside to rest herself. As she rose to pursue her way she inclined her body far forward. Her body, weighed down by the load, bent strongly forward and to the side. When the physician who treated the case arrived, he found the whole knee-joint much swollen, the leg strongly abducted. Overextension was readily produced and motion caused crepitus. He diagnosed the case as supracondyloid fracture, straightened the leg, and applied a plaster bandage. On removing the plaster, after several weeks, he was surprised by the unexpected state of affairs encountered, and called me in consultation. I found no sign of fracture, either on the femur or tibia, no shortening, but the leg was rotated upon the thigh and the joint almost completely stiff. There was a slight degree of equinus. The rotation was outward, so that a portion of the upper joint surface of the internal condyle of the tibia could be palpated in front of the internal condyle of the femur. Because overextension and abduction were possible immediately after the injury the diagnosis of the condition could still be made. In the preceding case the function of the knee-joint was completely restored; in this case partial anchylosis in slight rotation resulted. Therefore, it is important to recognise this injury.

At the time that Stark published two cases of rupture of the crucial ligaments the report attracted considerable attention. In both cases the following group of symptoms was found: After a fall producing hyperextension of the knee-joint, accompanied by audible cracking, the limb became helpless. The joint could not be steadied so as to permit of standing. In the sitting posture the knee could be actively and passively flexed and extended; at the same time the tibia could be displaced forward and backward on the femur. The noticeable fact in these cases was, that after the crucial ligaments had been ruptured no anterior dislocation of the tibia occurred. In parenthesis, we may mention that Stark's diagnosis was not entirely correct. If hyperextension and lateral mobility were present, the internal lateral ligament must also have been torn. The rupture of this ligament can be demonstrated by palpating the region occupied by the structure, with the leg flexed at right angles and rotated outward. If the ligament is torn a gap is noticed at the site at which its fibres are usually felt. In addition, we find that the leg can be slightly abducted, and that, with the knee flexed, a greater freedom of rotation can be demonstrated than on the healthy side. The external condyle, as has been previously mentioned, acts as the fulcrum. Rupture of the external lateral ligament I have never seen, but one case has been mentioned to me.

If any injury of the knee-joint is followed by excessive swelling, some difficulty may arise in the diagnosis. It has already been stated that rupture of a ligament can be readily overlooked. It is to be emphasized that sometimes even dislocations and fractures are hard to distinguish from one another without painstaking examination. This examination must necessarily demonstrate whether the constituents of the joint are in their normal relationship or not. If the tuberosity of the tibia occupies its normal relations to the head of the fibula and to the condyles of the femur, a dislocation may be excluded, no matter how equivocal the distortion of the axis of the limb may seem. The worst possible mistake would be to entirely overlook a dislocation or fracture hidden by excessive swelling, and to call the injury traumatic gonitis. If you are called upon to see such a case, examine thoroughly.

CHAPTER XXXIII

INFLAMMATORY SWELLINGS OF THE KNEE-JOINT

In dealing with *inflammatory processes* of the knee-joint, we will take a case of acute EFFUSION INTO THE JOINT as an example, and with this as a basis discuss the symptoms which arise and the method of proving the effusion to be intra-articular. If a hole is bored into the patella of a cadaver and fluid introduced into the joint through a cannula, a peculiar phenomenon is noticed. The patella is floated upward, the whole region about the knee-joint appears swollen, and the leg assumes a certain well-defined posture. The patella is merely raised by the fluid, which accumulates between it and the lower end of the femur. The position of the leg is due to the tension of a particular portion of the capsule. The outline of the distended joint indicates the extent of the synovial sac filled with fluid, and its shape is characteristic. In the normal state, the well-known grooves situated to either side of the patella can readily be seen; they disappear in the distended joint, and the outline of the patella simultaneously grows indistinct. Above the patella a swelling also appears, and extends upward upon the femur for two to three inches. The boundary of the swelling has its convexity directed upward. This increase in size above the patella owes its origin to a recess of the synovial membrane, which lies beneath the quadriceps tendon, and communicates below by an opening of large size with the main portion of the joint cavity. All these factors serve to give the neighbourhood of the knee-joint its rounded, spherical shape. During life the process is exactly identical when an acute effusion into the joint takes place. The extremity then assumes a position of moderate flexion, and the outline of the joint is of the above-mentioned variety. The spherical swelling shows fluctuation above and to either side of the patella, and this fluctuation is transmitted to all portions of the joint. The result is the so-called *dancing* patella (ballottement)—i. e., the patella may be depressed till it touches the femur, when it immediately rebounds.

A serous effusion into the knee-joint of traumatic origin is the result of severe sprains and contusions. Rheumatic gonitis occurs spontaneously, is accompanied by severe pain, and frequently by fever. Often other joints are similarly affected. If the pain is severe, and limited to the knee-joint, and especially if the joint is markedly rigid, *gonorrhoic gonitis* must not be forgotten. Therefore, examine the genitals.

Exudation into the knee-joint may, however, occur slowly and insidiously from various causes. In some cases the etiological factor is a mild, rheumatic infection, in others a tuberculous process sets in with symptoms of an exudative synovitis. Arthritis deformans may at the outset manifest itself merely by a slight effusion. If the individual is cursed with a tubercular family history, and if symptoms of tuberculosis are evident in other portions of the body, the prognosis of the case must be regarded as more serious—i. e., as a tubercular process—and in the further treatment this fact must be kept in view. An arthritis deformans will unmask itself during its further course by the grating sounds heard on motion, combined with thickening of the capsule, most noticeable at its upper margin. This trouble, although it causes but little discomfort, is also distinguished by its stubborn persistence. In a marked case, the capsule is enormously distended in the course of years; the grating sounds are loud and evident; pain is slight, and contractures fail to appear.

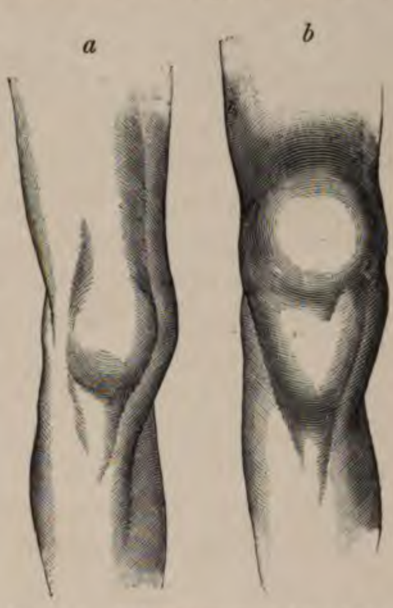

FIG. 52.—Anterior view of knee-joint: (*a*) in the normal condition; *b*, joint distended by large effusion.

Let us assume that the swelling unquestionably corresponds in its extent and outline to the joint capsule, but that floating patella is completely wanting. In this case the exudate can not be of fluid consistence. But, as the form of the swelling points to the capsule, we are not dealing with a distention by fluid, but a capsular swelling resulting from newly formed tissue increase. This is often due to TUBERCULOSIS. The process is characterized by a growth of young connective tissue about the capsule and granulation tissue which encroaches upon the joint. The capsule and ligaments are incorporated and replaced by this new material;

the cartilages and bones are invaded; tubercles are formed. Areas break down into pus, point, and discharge so that fistulous tracts lead into the joint. The outcome is the so-called caries of the knee-joint. After fistulæ have developed, crepitation, abnormal mobility, etc., can be demonstrated, and the condition is unmistakable. Before this stage, however, the condition should be recognised. The individual is of phthisical habitus; the knee can not be completely extended; its anterior surface is spherical and rounded, but fluctuation can not be distinctly obtained. The swelling has a soft, pasty consistence, due to the new-formed tissue; the skin is pale and somewhat shiny. Pain is variable, but abnormal lateral mobility is an early symptom. Absence of effusion in a joint which shows a rounded outline and lateral mobility, combined with the peculiar consistence just mentioned, as the chief symptoms, is typical of a destructive process, starting either in the epiphysis or, even more frequently, in the capsule.

In children, the primary focus is more often situated in the bone. The process may then heal without advancing to a suppurative stage. The knee-joint is swollen, but not in the same manner as when the joint cavity is distended. Next to the patella, and somewhat below it, are two small elastic welts. The upper recess is not distended, the patella does not float, for there is no effusion. The pathognomonic posture is assumed early in the disease, contractures result, and the muscles of the leg rapidly atrophy.

Sometimes the fungous proliferation is accompanied by a rapid intra-articular formation of pus—the so-called cold abscess.

Occasionally the beginner finds it difficult to determine the nature of swellings which appear at the knee-joint. Such are the *neoplastic tumours* of the lower end of the femur—sarcomata and osteochondromata. Myelogenous sarcomata are particularly apt to mislead, because they occasion severe pain in the knee, and produce an apparent loosening of the ligamentous structures. These symptoms appear to be due to an inflammatory process, especially the last-mentioned symptom, which seems to be a relaxation of the ligaments as a result of fungous processes. This laxity is only simulated. In extension the leg can be adducted and abducted more than the normal, but the motion takes place not in the knee-joint but in the sarcomatous mass, which has eroded the femur. Pain is not a reliable symptom. In the case of myelogenous tumours, it is readily understood that the pain may be severe and not diminished by rest. In order not to go wrong in such a case, other signs must be taken into consideration. Most important is the fact that in these new growths the characteristic position of flexion, which is never wanting in painful inflammatory processes, does not appear. The swelling produced by the bone tumours has a peculiar consistence. In none of the above-mentioned exudative processes (including among them the fungous invasion of the capsule) was the swelling hard; in sarcomata, isolated spots are softer, but others are positively hard. Myelogenous sarcomata may show parchment crackling in spots—a symptom due to a thinned-out, bony lamella covering the tumour mass. If present, it removes all doubts.

Just as in all other joints, it must not be forgotten that an acute swelling of the knee-joint may be the first symptom of an idiopathic *osteomyelitis* of the lower end of the femur or upper end of the tibia. The high fever, typhoid state, widespread œdema, the severe, boring pain, and especially the fact that particular spots on the bone are most tender to pressure, serve to distinguish the condition from acute articular rheumatism. When the symptoms of pus formation are noticed our last doubts are dispelled, for in rheumatism the process never advances to suppuration.

A striking picture is afforded by the arthritis sometimes seen in the course of *tabes*. The joint is much swollen, the joint cavity distended by a massive exudate, the femur considerably thickened (in the prepared specimen showing bony deposits). This thickening may extend to the middle of the femur. In the joint, loud cracking and creaking may be elicited, as if large pieces of bone lay free within the cavity. The joint has lost all of its firmness; it is loose, and at times shows a truly surprising lateral mobility. The patient uses the joint without a trace of pain. In addition, the usual signs of tabes are found.

It may be mentioned here that analogous changes also occur in the ankle-joint.

It ought no longer be difficult to diagnose *periarticular* swellings about the knee-joint. *Prepatellar bursitis* requires but casual mention. A strictly circumscribed, elastic, tense swelling directly in front of the patella is characteristic. A deep-seated *abscess in the popliteal space* is not as readily recognised. It may be due to suppuration of the subfascial tissues or to suppuration of a lymph gland, frequently found in this region. The joint is flexed, but *anteriorly* there is no exudation into the joint; the bones are not enlarged; tenderness is wanting. Only the popliteal space is fuller, tense,

d very sensitive. Soon the skin becomes dusky red,
d fluctuation can be obtained. In scrofulous children
d phthisical adults cold abscesses (extra-articular)
cur at the internal condyle. Their extra-articular
uation may be recognised by their circumscribed
ape, the normal contour of the rest of the region of
e knee-joint, and the absence of floating patella, in
ite of the fluctuation noticed over the abscess itself.

CHAPTER XXXIV

INFLAMMATORY SWELLINGS OF THE ANKLE-JOINT AND OF ITS VICINITY

IN no joint is the excellent method of injecting fluid, in order to reproduce a joint distended with exudate, of greater value than in the ankle. On the living this picture is rarely seen, and chronic effusions in particular are uncommon. On the other hand, as extra-articular inflammatory processes are of frequent occurrence, the picture furnished by distending the joint is of value in order to differentiate the two processes.

If it is impossible for you to see the ankle-joint distended, either in the cadaver or in the living patient, the following points will be of value: The ankle-joint distended by considerable effusion shows a distinct increase in its transverse diameter. This increase may be measured by means of calipers and expressed in figures. As the malleoli are only covered by skin at the points where the greatest transverse diameter of the joint intersects them, with the skin normal, any increase in diameter must be ascribed either to a swelling of the bones or to an increase of the joint contents. If examination of the bones shows no increase in their volume, no other explanation but that of a distention of the joint cavity remains. The outline of the swelling requires attention. The distended capsule protrudes at four points. Two prominences appear anteriorly, one to either side of the extensor tendons, which run in front of the joint, and hence must constrict the swelling; two much smaller protrusions appear posteriorly between the tendo Achillis and the external and internal malleoli. If the anterior prominences are compressed, the posterior grow tenser. The reverse manœuvre is not as successful, because compression of the flat posterior protrusions does not cause sufficient increase in tension; but it always is possible to demonstrate that these four points are all part of one continuous swelling. As the effusion tends to crowd the malleoli apart at the expense of the tibiofibular ligaments, which become more lax, it is possible in advanced cases to move the astragalus from side to side. This produces the so-called "bruit de choc malléolaire" due to pushing the astragalus suddenly to one or the other side, so that it strikes the joint surface of the malleolus.

Effusions into the tendon sheaths (tenosynovitis) about the ankle never can be confused with hydrops of the joint, for they all occur in the form of elongated swellings corresponding to the course of some tendon. On the anterior aspect of the joint their distribution is as follows:

The sheath of the tibialis-anticus tendon begins at least five centimetres above the malleolus, and ends about two centimetres below this point. A collection of fluid within this sheath produces a sausage-shaped swelling, which runs along the anterior edge of the tibia in a downward direction, over the extensor surface of the ankle-joint.

A similar effusion into the sheath of the extensor communis digitorum produces a broader swelling, which lies in front of and below the external malleolus. The swelling has another portion situated above the malleolus, separated from it by the annular ligament, but communicating with the lower one.

An hygroma of the synovial sheath of the extensor longus hallucis forms a narrow, slender swelling on the dorsum, extending down to the metacarpal bone of the big toe.

Posteriorly the following relations are observed: Behind the external malleolus is the sheath of the peronaei. Effusions into these produce an elongated swelling with its convexity directed backward. It extends six centimetres above the tip of the external malleolus, along the posterior surface of the fibula, conforming closely to the shape of the external malleolus, and finally, bending at an angle, runs forward along the external surface of the os calcis. Behind the internal malleolus we find the sheaths of the tibialis posticus, flexor communis digitorum, and, still closer to the Achillis tendon, the flexor longus hallucis. Effusions here must produce an elongated swelling between the inner edge of the posterior surface of the tibia and the tendo Achillis.

Effusions characterized by four elevations—two anteriorly and two smaller ones posteriorly—lead to examination of the joint. A single swelling anteriorly, close to the tibia, requires examination of the tibialis anticus; anteriorly, close to the fibula, to examination of the extensor communis digitorum; a swelling behind the lower end of the fibula requires examination of the peronaei; and finally, if behind the tibia, examination of the flexors.

I have seen well-informed students puzzled by the appearance of two swellings along the posterior surface of the joint, to either side of the tendo Achillis between it and the respective malleoli—i. e., one swelling behind the tibia and one behind the fibula, with the tendo between them. It should be borne in mind that tuberculosis of the calcaneo-astragaloid joint produces this picture. The pus points upward along the sides of the Achillis tendon; a cold abscess forms, and later fistulous tracts develop. Here we may add that the joint assumes a position of rest, and hence the foot is held in slight supination. This last symptom does not hold good if the tubercular process is situated along the superior surface of the os calcis immediately posterior to the joint. Nothing but the above-mentioned swellings to either side of the tendo Achillis and a crowding backward of this tendon results. The usual downward course of the tendon, with a slight concavity directed backward, is lost and changed either into a straight line or a curved line, with its convexity directed backward.

If the tuberculosis occurs on the lateral aspects of the os calcis, flat abscesses of small extent form on the surface of the bone, discharge externally, and produce one or more small fistulæ, which lead to softened bone. The whole neighbourhood is always much infiltrated and swollen.

Let us analyze the following findings: "The neighbourhood of the ankle-joint markedly swollen, collateral œdema on the dorsum extending along the lower third of the leg; in front of the external and internal malleoli a hot, fluctuating swelling covered with reddened skin, each swelling about the size of a pigeon's egg. Fluctuation was transmitted from one swelling into the other, but between the two the tissues were not crowded forward; behind the malleoli and along their exposed surfaces only marked œdema. Pressure of the heel upward produced no pain and no increase in tension in the tumours." As the fluctuating area was separated into two parts by the extensor tendons, the pus lay behind these tendons. As no fluctuating spots appeared behind the joint (merely œdema), as pressing the heel upward did not cause pain or increase the tension in the swellings, the pus could not be within the joint. Hence the pus was behind the tendons and in front of the joint. Diagnosis: phlegmon periarticularis anterior. After the swellings had been opened the exploring finger could be inserted behind the tendons, where it came in contact with the capsule. Water injected by one opening ran from the other. The connective-tissue layers in front of the joint had suppurated, and, on account of the great tension exerted by the tendons, had pointed along their sides. This accounted for the marked œdema. The process which developed in the course of six days completely healed in three weeks.

CHAPTER XXXV

INJURIES OF THE FOOT

THE diagnosis of effusions into the ankle-joint was discussed in the preceding chapter, because the question frequently arises whether a traumatic effusion into the joint has or has not followed upon an injury to this region. This question is most often asked when contusions or sprains have been sustained. The main points to be considered have just been dealt with.

On account of the frequency of injuries in this region, on the one hand, and common occurrence of tuberculosis of the tarsus on the other, it is well for beginners to give a guarded prognosis. This applies especially in cases of anæmic children, or those of tuberculous families, even if a positive history of trauma (falls, sprains) has been obtained, for tuberculosis may subsequently develop.

Acute osteomyelitis frequently follows subcutaneous trauma. Severe pain, persisting in spite of complete rest in bed, strongly marked œdema, and especially violent fever and prostration, indicate, from the very outset, that the process is serious. Red streaks appearing along the lymphatics without preceding injury to the skin, in conjunction with extensive œdema and fever, will at once point to a deep-seated suppurative process. Carefully conducted palpation of the tibia and fibula will lead to the discovery of some particularly painful spot in the neighbourhood of the inferior portion of the diaphysis. The exquisite tenderness of a part of the bone must fall heavily in the scales against acute articular rheumatism in cases in which the osteomyelitis has caused a joint effusion. In the course of a few days a thin layer of fluid collects over the maximum point of bone tenderness—corresponding to the development of a periosteal abscess.

In the foot, simple bone and joint injuries are very varied, owing to the manifold articulations met with. Isolated varieties occur, however, with varying frequency. While fractures of the ankle are of every-day occurrence, dislocations of the foot are extremely infrequent. The so-called fracture-dislocations, by which is meant the displacement of the foot due to Pott's fracture, are, of course, not included, for they are by no means rare.

Only two uncomplicated DISLOCATIONS are met with in the tibio-tarsal articulation—in which the tibia is either displaced in front of or behind the astragalus. If the anterior ligaments and capsule are torn by excessive plantar flexion, the tibia is displaced forward and protrudes through the tear. The foot remains in plantar flexion; the heel appears more prominent, the Achillis tendon is very concave posteriorly, and the anterior surface of the foot is strikingly shortened. The opposite holds good when the posterior portion of the capsule is torn by exaggerated dorsal flexion. The tibia then glides backward through the rent in the capsule, the foot remains in dorsal flexion, the heel disappears, and the anterior portion of the foot is markedly increased in length. Careful palpation will allow the displacement of the bones to be more accurately determined.

A similar, but less pronounced, sagittal displacement of the ankle in relation to the foot may take place if both malleoli are transversely fractured. The treatment of these fractures must be conducted in such a fashion that no abnormal lengthening or shortening of the heel remains. Correct reposition can readily be obtained if the knee-joint is flexed. I can not overemphasize this point, for skiagrams show how readily physicians neglect to correct minor degrees of displacement of the astragalus. But at the present time the lay public has, in the Röntgen ray, a means of controlling the physician.

Lateral dislocation of the ankle can take place only after one or the other malleolus has been fractured. The condition, therefore, receives the name of *fracture dislocation*. Internal displacement is uncommon; displacement outward is more frequent. We talk of external luxation when the foot is twisted in such a way that the inner edge of the sole lies at a lower, the outer edge at a higher, level, so that an extreme position of valgus results. The picture is striking. In addition to the tilting of the foot, the observer's attention is attracted particularly by the sharp protrusion of the lower end of the tibia. This part of the bone threatens at each moment to pierce the skin, and sometimes does so, particularly if the internal malleolus is torn off, so that the fractured surface of the tibia protrudes externally, producing a compound fracture. Frequently the patient is carried into the dispensary after some of the onlookers have reduced the displacement —workmen and farmers are especially skilful. The foot, however, retains the tendency to redislocate. Dupuytren has taught us to treat these fractures by placing them in the varus position, and thus counteracting the deformity. If this tendency is not overcome, healing of the Pott's fracture takes place in the valgus position, and the foot always remains less serviceable.

If a suspected malleolar fracture is not complicated by displacement of the foot, the extensive swelling interferes with the detection of the site of fracture. In order to save the patient much pain—certainly one of our duties—and yet to make the diagnosis positive at the first examination, it must be borne in mind that malleolar fractures occur at certain typical locations. The internal malleolus is fractured near its tip or its summit is torn off. This portion of the malleolus should therefore be palpated for a sharp, fractured surface. The external malleolus has not only its tip torn off, but transverse fractures also result. These, as a rule, take place above the tibio-fibular ligament—that is, immediately above the ankle-joint. The lower end of the fibula requires examination in a patient who, after a fall, can no longer step on his foot, and whose ankle-joint is obscured by the swelling. If an account of the relative situation of the foot and of the body at the time of injury can be elicited, the mechanism and the variety of fracture can usually be guessed at. Strong plantar flexion may tear off the summit of one or both malleoli; exaggerated dorsal flexion fractures the internal malleolus at the level of the articular surface of the tibia. Strong supination may tear off the tip of the external malleolus; strong pronation, the tip of the inner malleolus. Such simple directions of force rarely occur; as a rule, more complicated factors are met with. Strong adduction of the foot, with simultaneous supination of the whole foot, may lead to fracture of the fibula (even above the level of the joint); strong abduction, in combination with pronation, may cause a tearing fracture of the internal malleolus, immediately followed by fracture of the external.

The following infrequent forms of injury require brief mention:

That remarkable bone, the astragalus, which articulates with four other bones and has no tendon inserted into it, may be torn from its complicated relations and be displaced forward or backward. It may turn, or even tear through the soft parts, and be found lying by the side of the injured subject. In this case the diagnosis is not difficult. But, even in the cases in which the soft parts are not injured, the astragalus may be recognised by its outline, whether it is displaced backward and causes the Achillis tendon to arch, or whether it comes to lie upon the dorsum of the foot. In each and every case, the lower extremity is shortened, the ankle not fractured, and careful palpation of the protruding bone will serve to identify the astragalus.

By *Subastragaloid dislocation* is meant a dislocation in which the leg and astragalus form the upper, the rest of the foot the lower, dislocated member. The displacement may occur in a forward, backward, external or internal direction. The position of the foot defines the variety.

In the *internal* (mesial), the foot remains in marked supination. The head of the astragalus protrudes on the dorsum as a readily recognised eminence. The malleoli, so to speak, grasp it.

In the *external* (lateral), the foot is found in pronation. Along the inner edge of the sole, immediately beneath the internal malleolus, a considerable portion of the astragalus may be felt. The bone is between the malleoli.

In the *posterior*, the foot is held in equinus, the heel is prominent, and the anterior portion of the foot shortened. Upon the dorsum the protruding head of the astragalus is felt, although it retains its normal relationship to the malleoli.

In the *anterior*, the foot is in the calcaneus position, the heel has disappeared, the anterior portion of the foot is increased in length, and the astragalus palpable posteriorly.

In all four cases the relative position of the tuberosity of the navicular bone to the tip of the internal malleolus is changed according to the direction in which the displacement takes place.

CHAPTER XXXVI

REMARKS ON DIVERSE DISEASES OF THE FOOT

Many years ago there was a well-known "foot doctor" in Vienna to whom all patients suffering with diseases of the foot wended their way. Some, however, he was unable to relieve, so that occasionally a patient drifted into the clinic or into a private office. Frequently *flatfoot* was not recognised by this celebrated "specialist," hence a few remarks upon the subject of flatfoot.

According to an old saying, an ideally shaped foot should have an arch in which a bird can hide. But there are individuals, and also races, in whom the foot is quite flat, so that the sole leaves a complete imprint on a soft surface. By these footprints fugitive negro slaves were tracked, for their feet are devoid of any arch. Such feet, in contradistinction to *acquired* flatfoot, should be called *pedes plani*. The acquired form of flatfoot is known as *pes valgus*.

This state of valgus may also be called pronation, for the pes valgus is a pathologically pronated foot. The deformity causes much discomfort and disability, which is limited to the time when the patient stands or walks. The more advanced the disease, the more marked is the pain during both walking and standing. When the patient sits or lies down the pain usually ceases. If the famous "foot doctor" had only known this! The pain is situated beneath the internal malleolus, or on the dorsum in front of the ankle-joint, and in advanced cases also beneath the external malleolus. This pain is so characteristic of simple flatfoot, that if it continues after the patient sits or lies down, the deformity can no longer be classed as simple flatfoot. The condition is then complicated by rheumatism, arthritis deformans, or gout.

Pronation, and not sinking down of the arch, is the true cause of flatfoot. Practically, almost every valgus is also a pes planus, but, exceptionally, cases occur in which the characteristic pain—both in regard to situation and occurrence upon standing—is present without flattening of the arch. The arch is preserved, but pronation is marked. In these cases, however, the history is one of foot strain from overburdening. Later, when flattening appears, the deformity is typical.

All the foregoing applies to the static flatfoot. The rachitic flatfoot of children very rarely causes pain. Occasionally flatfoot results from rheumatism of the tarsus. In these cases an exudate may be demonstrated. Sometimes static flatfoot makes its appearance in later life (especially in women) if the individual shows a rapid increase in weight. If all these factors are kept in mind, it will save us from wandering blindly in the diagnostic labyrinth of gout, rheumatism, and neurasthenia. The shape of the foot, the characteristic localization of the pain, and its appearance upon standing are pathognomonic.

It would seem impossible to confuse clubfoot and flatfoot. And yet, in a work upon diseases of the foot published in 1895, by two respected English orthopædists, an illustration representing compensatory varus appears with the explanation that the *valgus* is due to relaxation of the ligaments. This is no printer's error, but it is—*fin de siècle.* A varus is wrongly called valgus—and why? The individual portrayed is suffering with genu valgum, consequently the foot is considered pes valgus.

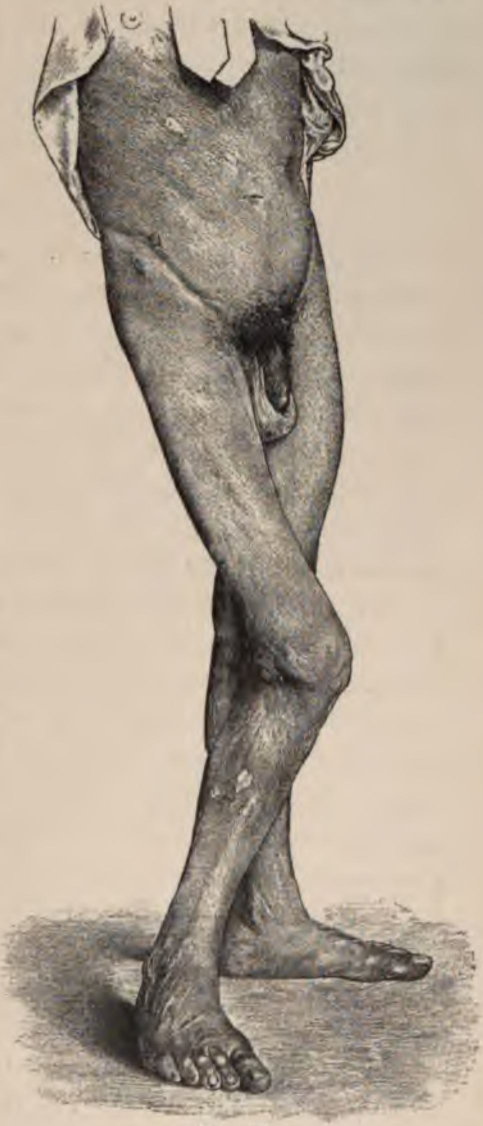

Fig. 53.

Let us analyze the case. In what manner must a person suffering with genua valga—i. e., a person with diverging legs—walk in order to bring his feet in normal relation to the tibiæ? He would have to walk on the inner edge of his foot. In order to become plantigrad he must supinate the foot, and supination equals varus. Conversely, a bow-legged person —i. e., a person with converging tibiæ—would have to walk upon the outer edges of his feet, unless some correction took place. This consists in pronation of the foot, so that the whole plantar surface can be placed upon the ground. A genu valgum hence produces a compensatory pes varus (recognised by the supination, marked arching of the dorsum of the foot, and adduction of the metatarsus); a genu varum produces a compensatory pes valgus (pronation, flattening, and absence of adduction of the metatarsus). Fig. 53 illustrates such a compensatory varus with right-sided genu valgum. The left knee is normal, but the left foot is, merely by chance, in a valgus position, which is recognised by the fact that the entire inner edge is placed upon the floor.

Of the various paralytic deformities of the foot I will emphasize one, because it is so rarely understood. It is the form described by Nicoladoni as paralytic calcaneus (pes calcaneus paralyticus). If well marked, it closely resembles the foot of a Chinese lady—short, with high arch. The arch of the foot is exaggerated to such a degree that the sole resembles a roof with transverse ridge-pole, so markedly is the anterior portion of the foot bent upon the posterior. The heel no longer points backward, but downward. The gastrocnemius and soleus are paralyzed, the peronæi functionate.

Formerly a condition, which I was then unable to explain, puzzled me greatly. Cases are met with in which one or both heels are painful, and walking much impaired. To either side of the insertion of the Achillis tendon a slight swelling, painful on pressure, may be observed. I named the condition *Achillodynia*. Professor Schüller expressed the opinion that the condition was due to inflammation of the bursa which lies between the tendo and the tuberosity of the os calcis. I agreed with him as soon as I had seen a case in which the effusion occurred in the course of a single night. Dr. Rössler, a student of mine, demonstrated anatomically that Achillodynia was a true Achillo-bursitis. The cause may be trauma, rheumatism, gout, or tuberculosis. But, before all other causes, do not forget gonorrhœa.

CHAPTER XXXVII

INJURIES OF THE VERTEBRAL COLUMN

We have repeatedly touched upon isolated diseases of the vertebral column. I recall to your attention dislocation of the cervical vertebræ, cervical Pott's, retropharyngeal and retro-esophageal abscesses, psoas abscesses, etc. There now remains only a general survey of the diseases of the vertebral column.

It is well known that the vertebral column is composed of an extremely complicated system of joints, and consequently it would seem fair to suppose that *traumata* would cause extremely varied injuries. The peculiar kind of joints met with, however, account for the fact that this expectation is not fulfilled, and physicians, from the time of Hippocrates down, have doubted whether a simple dislocation ever took place. Observations have since taught us that simple traumatic dislocation of the vertebræ may occur, and if it does, the site is in the cervical spine. Blasius has collected eighty-one cases of undoubted dislocation of the cervical vertebræ from literature, in which it is found that the dislocation most frequently takes place between the fifth and sixth cervical. It has been noticed that these two bones are also most often the seat of fracture.

The most striking symptom of dislocation of the cervical spine is the pathognomonic position of the head, which has been fully described—including both the symptomatology and the varieties of dislocation—in a previous chapter.

In the thoracic and lumbar portion of the spine dislocation never occurs without fracture, at least of the articular processes. The statistics collected by Gurlt show that in addition to the two above-mentioned cervical vertebræ, the fifth and sixth, the last dorsal and first lumbar vertebræ are most frequently injured. The symptoms of these fractures vary greatly. Simple *fissures* and *incomplete fractures* are not open to direct examination in the living subject; on the other hand, they produce no threatening symptoms. More serious are the *compression fractures* described by Middeldorpf, which consist of complete crushing, or severe bruising, of the spongy portion of the bodies of the vertebræ. These injuries may narrow the lumen of the spinal canal and crush the spinal cord. The severest type of injury is *fracture combined with dislocation*. Either the body is fractured (most frequent in the dorsal or lumbar spine, in which case the upper fragment is dislocated forward) or the lamina is broken, usually seen in the cervical spine. In the latter case one of the fragments is displaced. In both forms narrowing of the canal and compression of the spinal cord result, which alone or in conjunction with the extravasation produce paralysis. The disability is of varying extent and distribution. The *extent* of the paralysis, of course, affects all the nerves which take their origin below the site of injury. The upper limit, therefore, is formed by a line drawn about the thorax or abdomen, which corresponds to the peripheral distribution of the set of spinal nerves situated immediately above the injury. The higher the situation of the lesion the greater is the extent of the paralysis. The following is a more detailed description:

Fractures of the lumbar spine, passing through or below the third lumbar vertebræ, ordinarily produce no paralysis if the dislocation is not extreme. This is due to the fact that the spinal canal at this level incloses only the cauda equina, composed of strong and freely movable nerve fibres, which readily escape compression.

Fractures situated between the third dorsal and third lumbar vertebræ injure the spinal cord below the origin of the brachial plexus. The arms, therefore, escape, but the *lower extremities*, the *bladder*, and the *rectum* are paralyzed. Retention of urine and fæces results; later, when the sphincters of the bladder and rectum no longer can control the accumulated contents of their respective viscera, incontinence of urine and fæces develops. If the fracture is situated at a higher level, the abdominal muscles are paralyzed. This is promptly followed by tympanites, consequent crowding upward of the diaphragm, and impairment of respiration. Paralysis of the abdominal muscles especially impairs the phase of expiration. Injury still higher up may paralyze some of the intercostal muscles, thus further increasing the respiratory difficulty.

Fractures which pass above the level of the third dorsal vertebræ cause more extensive paralyses. Their great practical importance requires a still more detailed description. The brachial plexus is formed by the fifth to eighth cervical and first dorsal nerves.

Fractures below the fourth cervical will, therefore, *paralyze* the *upper extremities.* But, as all the intercostal and abdominal muscles are also paralyzed, so-called *diaphragmatic respiration* results. All the respiratory muscles, with the exception of isolated muscles of the neck, are now functionless; the diaphragm alone performs the act of inspiration. Expiration is carried on solely by the mechanical forces, of which the elasticity of the thorax is the chief factor. The immediate result of this disability shows itself in that all actions requiring an active expiratory effort, especially the acts of coughing and sneezing, are almost completely abolished. The condition becomes still more serious if bronchitis is present. The main fibres of the phrenic nerve pass out through the intervertebral foramen formed by the third and fourth cervical vertebræ. If a fracture involves these parts the phrenic may be injured, causing instantaneous death, or death shortly after the accident. Gurlt's statistics show that this fatal outcome has in some cases been due not to the fracture itself, but to passive movements undertaken at a later period. One patient's death was due to the fact that his daughter clasped her arms about his neck; another, because his wife placed her hand beneath his head to support it; a third died instantaneously when the barber turned his head while shaving him.

Fractures of the first and second cervical vertebræ cause instant death if the primary dislocation is of sufficient extent to crush the spinal cord. If the primary displacement is less marked, the patient may continue to live for days, until some sudden movement brings about the catastrophe. We know of one case of fracture of the atlas, without dangerous displacement, in which life was prolonged for a considerable period of time.

The completeness of the paralysis varies. As a rule, although motor symptoms are well marked, the sensibility of the skin is entirely unimpaired or only slightly affected. It must be emphasized that fractures in the region of the origin of the brachial plexus may cause complete paralysis of the lower portion of the body, and yet the upper extremities may escape all injury, or, at best, merely suffer incomplete pareses, for instance, of one arm or both forearms. In addition, it should be mentioned that in some cases reflex movements are almost completely preserved, so that tickling of the sole may produce movement of the toes in the paralyzed limb.

Paralyses have been mentioned before other symptoms, as they are most striking and important. The extent of the lesion furnishes some clew to the nature of the injury. In lateral displacement of the fragments, one side of the body suffers greater disability than the other, etc. We must assume, however, that the dislocation alone produces the paralysis. This will hold good in the majority of instances, but in isolated cases it may be next to impossible to exclude an extravasation as the partial cause of compression.

In addition to paralyses, *symptoms of irritation* may be present—hyperæsthesiæ, neuralgic pains, tonic and clonic spasms, and fibrillary twitching of the skeletal muscles. Fractures in the region of lower cervical and upper dorsal vertebræ may produce mydriasis or narrowing and immobility of the pupil, of which I saw an instance in the department of Professor Mosetig in Vienna. This phenomenon is explained by the fact that the nerve centre which controls the size of the pupil lies in this part of the cord, and is therefore exposed to irritation.

Vaso-motor disturbances are at times of special interest. In addition to blanching or reddening of certain regions of the skin, excessive rise in body temperature is sometimes observed after injury to the cervical part of the spine. Observations made on such cases have led to much (and as yet by no means complete) investigation upon the control of the central nervous system over the body temperature. Of similar interest is a case in which the sweat excretion suffered local impairment, for, though the lower extremities remained dry, the upper, uninjured portion of the body was covered with drops of sweat. Of theoretical importance is the ejaculation of semen which may take place immediately after an injury in the cervical region. Erections may occur shortly after the accident, and persist for days, either strongly marked or partial. These erections may regularly reappear after each catheterization, although the urethra is insensitive. Olivier, and later Goltz, have attempted to discover the physiological significance of this phenomenon by experiments performed on animals.

We have therefore a considerable number of symptoms which may accompany fracture of the spine. In some cases the diagnosis may, however, be difficult. The diagnosis is easy in those cases in which crepitus can be elicited, or where striking deformity of the spinal column can be demonstrated by palpation along the posterior surface of the body (or in injury of the cervical spine by examination through the mouth). Only in the cervical region will there remain a doubt whether dislocation is simple or complicated by fracture. If the deformity is slight it may be difficult to decide, in spite of palsies, whether we are dealing with a fracture or merely with compression due to hemorrhagic effusion. In many cases a positive diagnosis is not possible.

CHAPTER XXXVIII

TUBERCULOSIS OF THE VERTEBRAL COLUMN

TUBERCULOSIS of the vertebræ occurs only in certain well-defined portions of the bones. In the cervical segment the disease is in some cases situated in the articular processes; as a rule, however, the bodies, and, still more definitely, the anterior surface of the bodies, are affected. In children the erosion of the vertebræ makes rapid progress; the corresponding spinous process becomes prominent early in the disease, causing what is known as the Pott's hump or angular kyphosis. As soon as this is present, no further doubt need be entertained about the nature of the trouble, for the kyphosis seen in rachitis is *arcuate*; the column is arched, with convexity directed backward, and a slight lateral curvature is usually present. In Pott's, in addition to the painful, prominent vertebræ, the symptoms of a gravitation abscess may often be found. Such a symptom-complex is unmistakable. In the initial stages an expert may fail to recognise the disease. I therefore emphasize that the most trivial symptom must receive due attention. The mother usually draws our notice to the first symptoms. She relates that the child now maintains a strained, uncertain posture, and that the gait has become staggering. Turning or sitting grows difficult; the child sits down carefully and slowly, without bending its back. On attempting to pick up some object from the floor, the patient holds his spine rigidly. Approaching close to the object, and flexing both hips and knees, the child squats down with its back held straight, and picks up the object from the side. The condition is one of muscular rigidity of the spinal column, analogous to the fixation of the hip-joint in coxitis. The child avoids jarring of the body, and refuses to jump or run. It must not be forgotten, however, that in some cases both the rigidity and precautions against jarring are not prominent symptoms. Occasionally children with angular gibbus jump about and appear so active that a spondylitis is scarcely suspected. The other signs in these cases, therefore, require special attention. Sometimes from the onset, if the site of the lesion is in the dorsal segment of the spine, unilateral or bilateral intercostal pain or paræsthesiæ are found. Such patients have girdle sensations about the chest or a feeling of oppression in the epigastrium. Not to examine the spine, if these symptoms are present, would constitute no slight degree of carelessness. Palpation of the spines of the vertebræ will lead to the discovery of one which is exquisitely *tender* to pressure. Copeland recommends that a warm sponge be passed along the back; when the sponge passes over the inflamed vertebræ it is said to cause pain. Rosenthal passed the electrodes of a constant current, keeping them close to one another, along the spinous processes. At the diseased spot this manœuvre produced burning pain. If weakness of the extremities is already well marked at this period of the disease, it frequently happens that the weakness at once disappears if the superincumbent weight is removed from the diseased part by supporting the patient from the axillæ. This, of course, applies only to disease of the dorsal or lumbar spine. Early occurrence of contracture in some joint, for instance at the hip, may mislead, and cause us to suspect coxitis. The hip-joint is found flexed, extension is resisted, and the weakness of the extremities, to which attention is called by the parents, is assumed to cause a voluntary limp. I have seen an excellent surgeon fall into this error, therefore never omit to examine the spine in all cases of beginning hip disease. To mistake the diagnosis in a single case is more aggravating than to make many apparently superfluous and useless examinations. If increasing but gentle force serves to overcome the contracture without causing much pain, and yet the deformity soon recurs, coxitis may be ruled out. In the further course of the disease other symptoms arise which make it impossible to overlook the affection. In order to judge the stages of the disease properly, the general condition of the patient and the following three points must be kept in mind:

1. The local *deformity*. In discussing abnormal positions of the head, we have already drawn attention to the fact that when the trouble is located in the bodies of the vertebræ, kyphosis makes its appearance very early. It appears especially early in the dorsal segment, where the normal spinal column has a kyphotic curve from the time at which the child begins to walk to advanced age, in which a marked bow-like curvature —the senile gibbus—develops. Here the gibbus—the projecting spinous process—is most readily demonstrated. In the concave segments—in the cervical and

lumbar portions—the projecting spine of the vertebra is more difficult to discover. In the neck, especially in disease of the lower cervical vertebræ, a visible kyphosis develops. If torsion (rotatory deformity) is an early symptom, it speaks in favour of a focus of disease in the superior or inferior articular processes.

2. *Gravitation abscess.* We have mentioned, in discussing caries of the bodies of the cervical vertebræ, that retropharyngeal abscess may occur and lead to dysphagia and later to impairment of respiration. The difficulty experienced in swallowing is extremely troublesome, especially if a continuous desire to swallow is produced by the irritating action of the tumour. If a retropharyngeal abscess exists, it is safe to assume that the focus is in the body of the vertebræ. If the articular processes are the seat of the trouble, the abscess develops laterally in the neck, and leads to well-marked lateral bending of the cervical segment—that is, to torsion, scoliosis, lateral kyphosis, etc. These remarks may be applied to the other portions of the spine, *mutatis mutandis.* A gravitation abscess which descends along the anterior surface of the bodies of the vertebræ indicates that the bodies are the seat of the disease. Just as retropharyngeal abscess results from caries of the upper cervical vertebræ, so retro-esophageal abscess may be due to caries of the lower cervical or upper dorsal vertebræ. More frequently the pus points farther down, forming narrow, tortuous tracts, without causing compression or stricture of the esophagus. The inflammatory thickening of the neighbouring tissues prevents the rupture of the abscess into the thoracic or abdominal cavities. The abscess, by following along the track of the large vessels, finally enters the pelvis,

and attracts attention as a pelvic abscess. Very exceptionally perforation, with resulting fatal pleuritis or peritonitis, takes place. Lambl has described a case of rupture into the trachea. We have observed a case in which the abscess ruptured into the intestine. In its course downward into the pelvis the abscess can scarcely ever be recognised by percussing the back, because it forms long, narrow, tortuous tracts, which fail to give physical signs. After entering the pelvis, the abscess can be more readily demonstrated. In some instances a sense of increased resistance can be obtained in one iliac fossa, or as soon as attempts at deep palpation are made the child holds the muscles of the affected side of the abdomen more tense than on the normal side. At other times a deep-seated, resistant mass may be felt. Percussion may give dulness, or fluctuation may be obtained. I never fail to make this examination in every child with kyphosis of inflammatory origin. It is a remarkable fact that such abscess may disappear during the summer months, especially after suitable treatment has been instituted, only to reappear during the winter. Puncture may give only pure sero-pus. If, in spite of the presence of such an abscess, the hip-joint is free from psoas contracture, we may assume, with much certainty, that the abscess has made its way along the subserous tissues. It is retroperitoneal, but does not involve the psoas. If signs of psoitis are present, the abscess lies beneath the iliac fascia. With but few exceptions, psoas abscess is due to caries of the lumbar vertebræ. The symptoms have been described elsewhere.

In the cervical region caries of the transverse processes, or more often of the joints between the ascending and descending articular processes, causes laterally situated abscesses in the neck. In the dorsal spine caries of the joints produces gravitation abscesses along the back. The pus works its way through the numerous layers of muscles, creating a narrow, tortuous passage, and finally points far from the original site. The distance traversed may be considerable, usually below and farther from the median line, so that at first sight the causal factor may not be recognised. However, the local point of tenderness, discovered by deep pressure alongside the spinous processes, the limitation or absence of all rotatory movements of the spinal column, and the normal condition of ribs, scapula, and ilium—for abscesses may point upward along the back from this bone—permit the diagnosis to be made.

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3. Symptoms indicating participation of the *spinal cord*, *meninges*, and *nerves*. Pathological anatomy has shown that the swelling of the inflamed portions of bone may involve the nerves, and thus give rise to the processes of neuritis and perineuritis. In the advanced stage the nerves appear atrophied, either as a result of a neuritis or secondary to degenerative changes in the cord. The meninges take part in the process as the result of inflammation by contiguity. Not only do thickening and clouding of the membranes occur, but the pus derived from the osseus inflammation spreads and peels the meninges away from the bone. The resulting abscesses may narrow the spinal canal to a considerable degree. The inflammation often involves the cord itself, so that myelitis is a not infrequent sequel. Soft, sometimes actually fluid, areas are found as a result of the myelitis. In the course of months, secondary degeneration of the tracts, both

upward and downward, occurs in the typical manner first discovered and described by L. Türk. Another frequent complication is compression of the cord due to various causes which have been previously indicated. How do these processes show themselves? Clinically, the neuritis manifests itself as neuralgia. The eccentric pain, intercostal neuralgia, and evident participation of the intercostal nerves characterizes the process, if in the dorsal region. The meningitis, as such, gives no symptoms. Involvement of the cord results in compression palsies. This is accounted for by the fact that, in addition to the myelitis, some compression is always caused either by bony displacement or by an abscess pointing into the canal. Paralysis due to compression is characterized by certain features: 1. The predominance of motor over sensory disturbances. 2. The great exaggeration of reflexes on the side subjected to irritation, unless the lesion is situated high up in the medulla, when the increase appears on the opposite side. Sometimes slight tickling of the sole of the foot promptly causes movement of the toes, foot, knee, and hip-joint on the paralyzed side, and, more rarely, erection of the penis. 3. The more or less complete disappearance of all the signs of paralysis where extension or a well-fitting corset is applied.

Brown-Séquard noticed that the paralysis disappeared after twenty-four hours of extension. V. Dumreicher presented the case of a blacksmith before the Vienna Society of Physicians, in which the patient, who previously was barely able to crawl about, at once walked several miles, after a well-fitting apparatus had been applied, in order to fetch the money demanded for the support. Of course the outcome is not invariably favourable. The sudden subsidence of the palsy seems magical on supporting the patient with both hands in the axillæ, thus raising the superincumbent weight from the inflamed parts. Such a successful experiment at once convinces both the patient and relatives that mechanical treatment is far superior to salves and other remedies.

With such a variety of symptoms to rely upon, it is evident that there should be no difficulty in making the diagnosis of spondylitis. If a distinctly anæmic or scrofulous child has an angular kyphosis, with local tenderness over the prominent spinous process, it is suffering with spondylitis. That is the whole diagnostic apparatus. The diagnosis can be difficult only at the onset, before deformity appears. After its appearance, disease of the vertebræ can no longer be doubted.

The course of spondylitis in adults differs materially from that in children. While the gibbus develops in the course of a few months in infancy, it may require years to develop in the adult. In adults the rigidity of the spine, pain or pressure over the spinous processes, and especially pain produced by jarring (for instance, driving over uneven cobble-stones), is especially striking. As the course of the disease is more insidious than in children, its diagnosis becomes more difficult. In spite of this fact, even in the early stages, it can be confused with but few diseases. One disease requiring differentiation from Pott's disease is the so-called *hysterical spine*, which is characterized by marked sensitiveness over one or more vertebræ, and by pain on moving the spinal column. This is one of the chief symptoms of *spinal irritation.* It is seen in nervous, hysterical persons. The lightest pressure upon the spinous process causes much pain; firmer pressure is less painful; immobility is slight, and the symptoms are inconstant. Another case might be characterized by neuralgic pains, girdle sensations (feeling of a constricting band), and later by the signs of a compression palsy. These may be due to circumscribed myelitis or tumours of the cord. In spondylitis, deformity must be well advanced before symptoms of compression appear. The neuralgic pains which precede the paralysis in diseases of the cord, as the first symptom, are unaccompanied by the rigidity of the spine, which is well marked in the early stages of spondylitis. At the onset, and even longer, the diagnosis may hover between caries and sarcoma of the vertebræ. The decision usually rests upon the great severity of the pain from the very outset in sarcoma, upon its persistence in spite of prolonged rest and proper treatment, and the failure of angular deformity to develop later on. Schlesinger mentions the following points in favour of the diagnosis of neoplasm: Bow-shaped kyphosis, *lateral* displacement of the spinous processes, tenderness to pressure next to the spine (without tenderness of the spinous processes), trophic skin disturbances, and herpes zoster.

In recent years several other diseases of the bones, which lead to distortion of the trunk, have been recognised.

Kümmel has described, as a special variety, a *traumatic spondylitis* in which there is a painful period following the trauma; subsequently a painless one, during which, in the course of months, a slowly progressing deformity of the trunk develops. In the majority of cases we probably are dealing with an unrecognised fracture of the vertebræ.

Paget's disease is the name applied to a certain variety of osteitis deformans. Its chief symptoms are the advanced age at which it makes its appearance, the bow-shaped kyphosis and rigidity of the spine, the slow, protracted course extending over many years, frequently starting with pain and deformity of the legs (position of supination and adduction), or the unsightly enlargement of the head, which is tilted forward.

Kahler's symptom-complex comprises the following: Severe distortion of the trunk (vertebral column, ribs, and sternum), great tenderness of certain spots, alternating with painless intervals, excretion of the Bence-Jones body in the urine. The suspicion that multiple myelomata always gave rise to this complex has not been verified.

In contrast to these rare forms we now mention the frequent picture of *osteomalacia,* which is well known from time immemorial: Onset during pregnancy or puerperium, exacerbation during succeeding pregnancies, changes in the bony pelvis, paresis of the ilio-psoas, adductor contracture, therapeutic influence of phosphorus.

CHAPTER XXXIX

SCOLIOSIS

In the domain of lateral deviation of the spinal column the physicians of our time are still guilty of many sins. On the one hand, beginning scoliosis often escapes unrecognised; on the other, slight lateral deviations are not taken seriously, and are passed over with the remark that they will disappear without treatment. The physicians should learn from the mothers—this includes both the recognition and the due appreciation of the gravity of the trouble. Scoliosis in a young girl is of the greatest significance, for it may endanger her life later on.

In dealing with a beginning scoliosis have the child stripped naked and place it upon a table or chair, in order to carefully observe it from the back. The child must stand in military posture, with its arms hanging down naturally and with the knee-joints fully extended. First of all, determine whether the legs are of equal length or not (the position of the gluteal folds will show this), for not infrequently scoliosis is purely of *static* origin. In these cases the deformity disappears as soon as the shortening is corrected by placing a book or piece of board beneath the foot of the shorter side. If the legs are of equal length, the relation of the arms to the trunk should be compared on both sides. If one arm is separated by a wider interval from the trunk, most probably the dorsal segment is more convex toward the opposite side. If the triangle at the waist (*Taillendreieck*)—i. e., the interval between the arm applied to the body and the lateral contour of the trunk—is not quite symmetrical, the lumbar spine is scoliotic. A considerable deviation may exist, and yet both of these signs be wanting. The disease is then recognised by the symptoms observed on the trunk. Test the position of the shoulder-blades. The mothers themselves point out this sign to the physician. If the one scapula is nearer to the spine than the other, asymmetry points to scoliotic deformity. Following up the spinous processes, we observe a slight deviation in the dorsal or lumbar segments. The contour of the thorax must also be examined. In the dorsal segment, when the case has progressed further, the posterior portion of the ribs shows an inequality in their curvature. A decided hump is formed by the ribs on the convex side of the curve. In the lumbar segment this difference manifests itself in the inequality of the sacro-lumbalis group, which forms a more decided prominence on one side than on the other. In order to recognise minor grades of rib deformity it is advisable to have the patient elevate the arms, or, still better, to have the arms placed in front of the body as if in the act of swimming. With the child in this position, it is a good plan to stand in front of it, and then ask the child to bend its head downward and forward. By this manœuvre we see the trunk from neck to pelvis in its reversed position, and are better able to recognise the slightest asymmetry between the right and left side. Also observe the junction of trunk and pelvis; if asymmetry is found, a scoliosis is probably present. This symptom has always been known as "*high hip.*" Finally, one must decide whether the physiological curvature of the dorsal segment is diminished. If this change has taken place, especially if the spine between the shoulder-blades is straight or concave instead of convex, an advanced degree of scoliosis is to be feared. If all these tests are made and nothing is observed, although there is a beginning scoliosis, the observer himself is a "hopeless case." If, on the other hand, he observes some deformity, it is his duty to recommend orthopædic treatment. *Initia morborum curantur!*

The scoliosis of infancy—not including static deformities—are due to rachitis. Scoliosis which appears in the school-girl is usually the result of habit, although late manifestations of rachitis may be at work. The dorsal segment is then convex toward the left, and the greatest deviation is found about the middle of the spinal column. The bend is quite abrupt and the spine very rigid. If other signs of rickets are present they strengthen the diagnosis.

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